FILE COPY
NOV 7 1957

HOME SAFETY Review

VOL. 14, NO. 8. OCTOBER 1957

To Johnny these may taste like candy.
We know aspirin shouldn't be so handy.

Before little Johnny breathes too much,
Tell him DDT means Don't Dare Touch.

Illustrations—
Virginia Health Bulletin
Virginia Dept. of Health

See that deadly bottle of bleach?
It shouldn't be within Susie's reach.

Steve drank that bottle in the medicine chest.
Guess we don't need to tell you the rest!

IN THIS ISSUE: Articles on poisoning control.

HOME SAFETY CONFERENCE

Chairman, JOHN C. THORNTON, chairman, Committee on Human Safety, American Institute of Architects, Royal Oak, Mich.

Chairman-elect, MRS. GEORGE WELLES, JR., St. Louis County (Minn.) Safety Council, Duluth, Minn.

Vice chairman, Community Service Groups Section, MISS MARY M. WEEKS, health and safety adviser, Girl Scouts of the U.S.A., New York, N. Y.

Vice chairman, Health and Medical Section, ROBERT H. KOTTE, M.D., American Academy of Pediatrics, Cincinnati, Ohio.

Acting vice chairman, Home Building and Equipment Section, E. J. HERRINGER, sanitary engineer, Region V, U. S. Public Health Service, Chicago, Ill.

Staff secretary, THOMAS FANSLER, director, Home Safety Division, National Safety Council, Chicago, Ill.

●

The Home Safety Conference is the meeting point for representatives of national organizations conducting programs that include prevention of accidents in the home. It officially represents National Safety Council home safety service subscriber-members. Conference officers (with the exception of the staff secretary) serve voluntarily without pay as the council's safety officers and planning board for home safety program.

National Safety Council
Chartered by the Congress of the United States

Reg. U.S. Pat. Off.

HOME OFFICE
425 North Michigan Avenue
Chicago 11, Ill.

EASTERN OFFICE
Room 733, Lincoln Building
60 East 42nd Street
New York 17, N. Y.

WESTERN OFFICE
Room 250, 703 Market Street
San Francisco 3, Calif.

HOME SAFETY REVIEW

A Magazine for Home Safety Program Leaders

VOL. 14 **NO. 8**

OCTOBER 1957

FEATURE ARTICLES

Who's Responsible for Poison Control? *Edward Press, M.D.* 3
Poison Control Centers on the Increase 4
A Poison Prevention Project, *Miss Edith R. Doane* 5
Incidence of Injury with Pesticides, *Bernard E. Conley, Ph.D.* 6
How Toxic Is It?, *Robert E. Gosselin, M.D., Ph.D.* 9
A 3-Year-Old Plays Nurse 13
Manufacturer Looks at Child Poisoning, *John H. Foulger, M.D.* 14
The Exposure of Children to Lead, *J. Julian Chisolm, Jr., M.D., and Harold E. Harrison, M.D.* 20
Practical Management of Common Poisoning, *Jay M. Arena, M.D.* ... 24
Inservice Training for the PHN, *Mrs. Mary K. Foster, M.P.H.* 26
Poison Antidote Chart 30

MRS. ELIZABETH REED, editor
GORDON O'NEILL, associate editor, home building and off-the-job
JOHN MUNROE, artist
NORVAL BURCH, editorial director, National Safety Council publications

●

The HOME SAFETY REVIEW is published monthly (except July and August) by the National Safety Council, 425 North Michigan Avenue, Chicago 11, Ill. Copyright 1957 by the National Safety Council. Printed in U.S.A. Price $.25 per copy, subscription $2.25 per year.
Statements and opinions advanced in signed articles are personal expressions of the authors, not necessarily those of the National Safety Council.

Chairman of the Trustees
W. S. S. RODGERS, former chairman, The Texas Co., New York, N. Y.

Chairman, Board of Directors
W. A. STEWART, trustee and former president, American Optical Co., Southbridge, Mass.

President
NED H. DEARBORN, Chicago, Ill.

Vice presidents:

For farms
KIRK FOX, editor, *Successful Farming*, Meredith Publishing Co., Des Moines, Iowa.

For finance, and treasurer
WILLIAM H. LOWE, treasurer, Inland Steel Co., Chicago, Ill.

For homes
GEORGE M. WHEATLEY, M.D., Metropolitan Life Insurance Co., New York, N.Y.

For industry
R. H. FERGUSON, assistant director, industrial relations, Republic Steel Corp., Cleveland, Ohio.

For labor
P. L. SIEMILLER, general vice president, International Association of Machinists, Chicago, Ill.

For local safety organizations
WALTER K. KOCH, president, Mountain States Telephone and Telegraph Co., Denver, Colo.

For membership
EDWARD C. MYERS, assistant vice president, United States Steel Corp., Pittsburgh, Pa.

For public information
BOYD LEWIS, vice president and executive editor, NEA Service, Inc., New York, N. Y.

For schools and colleges
LOWELL B. FISHER, Ph.D., state chairman, North Central Association of Colleges and Secondary Schools, Urbana, Ill.

For traffic and transportation
WALTER F. CAREY, president, Automobile Carriers, Inc., Flint, Mich.

For women's activities
MISS MARION E. MARTIN, commissioner of labor and industry, State of Maine, Augusta, Maine.

Executive vice president
G. C. STEWART, Chicago, Ill.

Secretary
R. L. FORNEY, Chicago, Ill.

Who's Responsible for Poison Control?

THE WORDS *poison* and *poisoning* often conjure up the specter of sudden and unexpected death. To many they have connotations only of suicide and murder, of intrigue and assassination. Too many persons still associate poisons with only a relatively few substances such as cyanide, arsenic, strychnine, and iodine. It is not until people are suddenly faced with a tragedy or, what is almost as bad, the torment of not knowing whether an accidentally swallowed common household substance is or is not poisonous that the full realization of ubiquitous distribution of potentially poisonous substances into almost every home hits them.

Modern technology has made thousands of chemical compounds available to lighten household chores, alleviate pain and disease, and help us in our everyday work and play. Like many other products of our inventive genius, when properly used, they are a boon to mankind; improperly used, by accident or intention, they may be harmful or even fatal. Unfortunately, they are improperly used many hundreds of times daily in the United States, and each time this occurs the specter of possible sudden death looms. Only little satisfaction can be obtained from the knowledge that statistics show that in only about one or two hundred such incidents does death actually strike.

The anxious parent and the perplexed physician may go through seemingly interminable minutes or hours until they know for sure whether the ingested substance is a relatively innocuous compound or a deadly poison or what the best and most modern treatment should be. It is this suspense over the outcome, caused partially by a lack of knowledge of the potentially toxic ingredients of many of the more than 250,000 trade name products distributed throughout the country, that has been a major factor in the enthusiastic reception of and amazingly rapid way in which poison control centers have caught hold and spread throughout the country.

Although the treatment of an acute poisoning episode may be more spectacular and dramatic than preventive efforts, in poison control activities the necessity for treatment spells the failure of prevention. Prevention is the keynote to control, and efforts at prevention should go hand-in-hand with treatment plans and procedures.

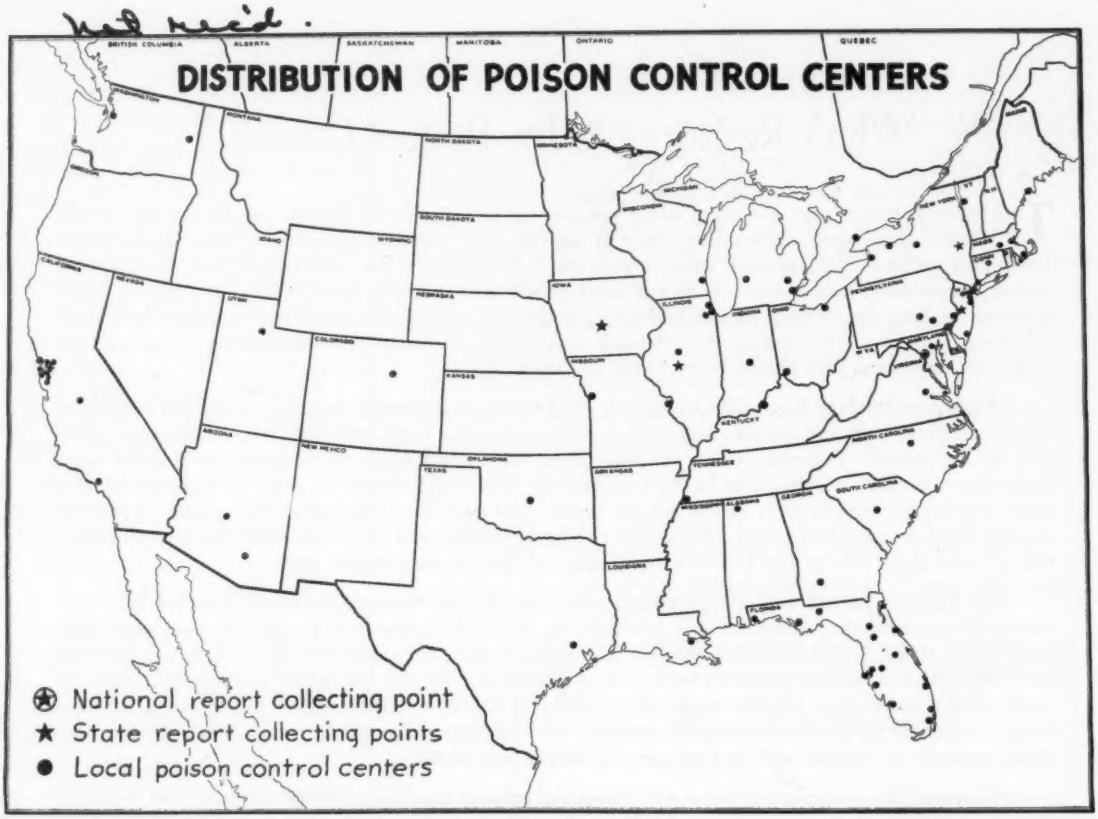
It is in this essential and widely based field of prevention that there is a role for all. It is not an activity limited only to doctors, nurses, and pharmacists. The well-organized and comprehensively operated poison control center encourages the participation of all relevant segments of the population. There is a role for the professional and for the general public, for industry and schools and universities (botanists, entomologists, and zoologists), for parents, teachers, rescue squad personnel, and for many others.

In effect, just as the fruits of the vast chemical industry are enjoyed by the community as a whole, so the community as a whole has a responsibility for seeing that these chemicals are properly used with the utmost in practical efforts to prevent poisoning.

—Edward Press, M.D.

Field Director
American Public Health Association
New York, N. Y.

POISON CONTROL CENTERS ON THE INCREASE


DISTRIBUTION OF POISON CONTROL CENTERS
National report collecting point
State report collecting points
Local poison control centers

SINCE THE inception of the first poison control center in Chicago by the American Academy of Pediatrics in 1953, approximately 90 poison control centers have developed in the United States and its Territories and Canada. Usually a hospital serves as the center, but such organizations as local medical societies, medical schools, and health departments are also centers. Physicians can refer to these centers for information on the composition of substances involved in poisonings and on methods of treatment, and they in turn can add to the knowledge gathered in the centers through their case experience.

In some states reports from all or nearly all local centers within the state are gathered by the state health department, and the Public Health Service in Washington, D. C., gathers reports on a nationwide basis.

Below is a list (prepared with the knowledge that was available September 1) of the cities indicated on the map above in which there are local poison control centers and in which there are state collecting points. Some of the state collecting points also have local poison control centers as can be seen by the list below.

STATE COLLECTING POINTS

California: Berkeley; **Florida:** Jacksonville; **Illinois:** Springfield; **Iowa:** Des Moines; **New Jersey:** Trenton; **New York:** Albany.

LOCAL POISON CONTROL CENTERS

Alabama: Florence; **Arizona:** Phoenix, Tucson; **California:** Berkeley, Fresno, Los Angeles, Martinez, Oakland, San Francisco, San Jose, San Leandro, San Mateo, San Pablo, San Rafael, Santa Clara (not shown on map above);

Colorado: Denver; **Connecticut:** Hartford, Stamford; **Delaware:** Wilmington; **District of Columbia:** Washington; **Florida:** Daytona Beach, Fort Lauderdale, Fort Myers, Gainesville, Jacksonville, Lakeland, Miami, Ocala, Orlando, Panama City, Pensacola, St. Petersburg, Sarasota, Tallahassee, Tampa, West Palm Beach;

Georgia: Albany; **Illinois:** Berwyn, Chicago, Evanston, Peoria, Springfield; **Indiana:** Indianapolis; **Iowa:** Des Moines; **Kentucky:** Louisville; **Louisiana:** New Orleans; **Maryland:** Baltimore; **Massachusetts:** Boston, New Bedford, Worcester; **Michigan:** Detroit, Grand Rapids, Pontiac;

Missouri: Kansas City, St. Louis; **New Jersey:** Atlantic City, Long Branch, Montclair, Newark, Nutley; **New York:** Albany, Buffalo, New York, Rochester, Syracuse; **North Carolina:** Durham; **Ohio:** Cincinnati, Cleveland; **Oklahoma:** Oklahoma City; **Pennsylvania:** Harrisburg, Lancaster, Philadelphia; **South Carolina:** Columbia; **Tennessee:** Memphis; **Texas:** Houston; **Utah:** Salt Lake City; **Vermont:** Burlington; **Virginia:** Richmond; **Washington:** Seattle (Bellevue), Spokane; **Wisconsin:** Milwaukee;

Hawaii: Honolulu (not shown on map above); **Ontario, Canada:** Toronto.

—Photo by Edw. Hering

A poison prevention poster project display provided by Mrs. Margaret Walter (right) of the Union County Safety Council, cosponsored by the Ladies Aux., Winfield Volunteer Fire Dept. Left is Lt. Norman Whitehouse, Winfield (N. J.) Fire Dept. and First Aid Squad.

A Poison Prevention Project

By Miss Edith R. Doane

Director
Women's Division
New Jersey State Safety Council
Newark, N. J.

ISN'T IT TRUE that one way to get volunteers to help with home safety programs is to give them something to do that will spark their interest so successfully that it can be fanned into concrete activity?

We of the New Jersey State Safety Council experimented with such a program last year and consider that it has been a very worthwhile activity, although its results are quite intangible at present. It has been a springboard to motivate a meeting or a continuing program and also has been used separately as a visual aid. We called it the Poison Prevention Poster Project.

The poster used in the project was adapted from the front cover of the May 1956 issue of the *Home Safety Review* which showed the two chief adult actions responsible for child poisoning: poor storage of poisons and lack of adult supervision. To this we added some positive educational material about poison prevention. The poster and the material were then mounted upon a wide, green border, easled, and used as the central background for an exhibit. A display of different shapes of empty containers that formerly held types of household products, medicines, etc., known to cause poisoning incidents when their contents were *misused,* was developed to accompany it. The containers were relabeled with such type labels as "silver polish," "insecticide," and "aspirin." The cost, always an important factor, was relatively moderate.

A letter with instructions on how to set up a display using the poster and empty containers was sent to every member of the State Exec. Com. of the Women's Div. of the N. J. State Safety Council and to all members of the county council committees and later furnished to individual groups that requested program aids. The actual arrangement was left to the imagination and resourcefulness of the sponsoring group.

Two copies of the poster were provided to each women's county safety committee, and two sets of containers, etc., were collected and stored ready for bookings. The following are typical of the many scores of showings that this "3-P" Project had during the program year and are indicative of the wide interest it created:

N. J. Cong. of Parents and Teachers; the fall conference of the N. J. Fed. of Women's Clubs and the annual meeting of its Junior Membership Dept.; department meetings of the Am. Legion Aux. of N. J. and the Ladies Aux., VFW; Ladies Aux. of the N. J. State Firemen's Association; PTA county council conferences, workshops, and local unit meetings; Women's Aux., Med. Soc. of N. J.; Homemakers' Day programs for the advisory council of the extension service; Child Study Groups of the N. J. Div., Am. Association of Univ. Women; and the Eleventh Annual State Home Safety Forum.

This type of display does not need to be attended, but it can be if the situation requires it. In addition, the accompanying exhibit material is readily obtainable in most homes, is easily stored in a box or two for transportation, and is simple to arrange at the exhibit spot. A minimum of space of about two card tables proved necessary. When too much more space is available, the tendency is to expand the exhibit with more items but not to increase its positive educational effects.

The "3-P" Project put a concrete program aid in the hands of safety chairmen. It enabled them to offer something with a fresh appeal on an old theme which could give them something special to highlight in their annual reports. It also provided opportunities for photographs, tie-in publicity, radio interviews, etc. In addition, with several scores of committee women actively concerned with collecting the exhibit items, labeling them, and setting them up, it was putting more positive thinking and action behind poison prevention in their own homes!

Incidence of Injury with Pesticides

By Bernard E. Conley, Ph.D.
Secretary
Committee on Pesticides and
Committee on Toxicology
American Medical Association
Chicago, Ill.

THIS PAPER was read in the Symposium on Childhood Poisons before the Joint Meeting of the Section on Pediatrics and the Section on Preventive and Industrial Medicine and Public Health at the 105th Annual Meeting of the American Medical Association, Chicago, Ill., June 12, 1956. The complete paper has been published in the *Journal of the American Medical Association*, vol. 163, No. 15, pp. 1338-1340, April 13, 1957.

THE PREVENTION of poisoning, whether by drugs, industrial chemicals, or pesticides, is rapidly becoming recognized as a leading health need. The pesticides (economic poisons) are an important element in this problem, although, as causes of poisoning, they have not always received the consideration that their record as causes of injury deserves.

Today chemicals for pest control are widely used in agriculture, in industry, in public health, and in and around the home. Over 200 basic chemicals in approximately 80,000 formulations have been registered under the Federal Insecticide, Fungicide, and Rodenticide Act. The basic compounds vary in their capacity to produce injury as is shown in table 1, which compares the safety of 81 common pesticides. The starred compounds are those from which fatalities have been reported.[1] [See page 8.]

The degree of risk involved in the usual or customary use of pesticides depends on a variety of factors other than the intrinsic toxicity of the chemical. The form (liquid, gas) and formulation (oil solution, wettable powder) of the product; its method of application (mist or coarse spray); frequency, amount, and duration of exposure; and other circumstances of use all influence the degree of risk. In regard to these factors, the following generalizations hold true.

Poisoning may occur both from single exposures to appreciable amounts of a pesticidal agent and from frequent, successive exposures to small quantities. The use of preparations containing solvents and wetting agents in the formulation or of finely dispersed preparations and the presence of oil or grease on the skin facilitates absorption of the toxic products into the body. Application under confined conditions such as in an unventilated room or lack of suitable protective equipment when the preparation is handled for prolonged periods or is in concentrated form also reduce the safety with which pesticides may be employed.

TABLE 1.—*Comparison of Safety of Pesticides*

Low Risk	Moderate Risk	High Risk
	Insecticides	
allethrin derris* methoxychlor p-dichlorobenzene Perthane propellant (Freon) pyrethrum* synergists	aldrin, BHC* calcium arsenate,* chlordane* chlorothion, DDT* Diazinon, dieldrin Diptherex, endrin* lead arsenate* Lethanes*, lindane* malathion, metaldehyde* naphthalene* pentachlorophenol* silicofluorides (sodium, mercury)* sodium fluoride* solvents (kerosene and others)* Thanite, toxaphene*	demeton (Systox)* hydrogen cyanide* Metacide* methyl bromide* nicotine* parathion* Paris green* schradan (OMPA) TEPP*
	Rodenticides	
Fumarine Pival red quill Tomorin warfarin	alpha naphthylthiourea-(ANTU) barium carbonate* zinc phosphide	arsenious acid* castrix* phosphorus (yellow)* sodium fluoroacetate* strychnine,* thallium*
	Fungicides	
Bordeaux mixture captan, chloranil copper carbonate ferbam, glyodin maneb, sulfur zineb, ziram	dichlone dichloroethyl ether* lime-sulfur* nabam pentachlorophenol* thiram	alkyl mercury salts* mercuric chloride* phenyl mercury salts
	Herbicides	
2,4-D* endothal maleic hydrazide monuron, diuron (chlorophenyl dimethylureas)	fuel oils* sodium borate* sodium chlorate* sodium trichloroacetate	dinitro-o-cresol* sodium arsenate* sodium arsenite sulfuric acid*

Types of Accidents

Injury from pesticides can arise from several sources: in agriculture, during application to crops or during harvesting of crops; in industry, during the manufacture or formulation of products; in public health work for the control of nuisance or disease-carrying insects; or in and around the home, either directly through application or indirectly through excessive residues on food or excessive atmospheric concentrations during crop dusting and community fogging operations. Needless to say, injury has been reported in all of these areas.

* * * * *

Incidence of Injury

The incidence of injury from pesticides has been extremely difficult to determine. Statistical data on nonfatal and fatal poisoning have been scanty and misleading, and

this has contributed to the misconception that pesticide injuries are infrequent or rare. In the absence of any ready and reliable index to the frequency of such injuries, resort is usually made to mortality statistics on accidental poisoning. As is evident from table 2, no detailed breakdown of the chemicals causing fatalities is provided, except for a few familiar industrial and medicinal chemicals. Even fewer (11) categories were provided before the sixth revision of the International Lists of Diseases and Causes of Death in 1948. It is, therefore, understandable that the belief should arise that the actuarial figures for pesticide injuries were so small as to be of only minor concern, especially when the data are compared with other causes of injury, such as drugs, petroleum solvents, and noxious gases.

Under current methods for recording fatalities due to poisoning, most compounds are classified in the "other and unspecified" category. A review of the substances hidden under this heading was undertaken for the Committee by the National Office of Vital Statistics.[8] The results of this study are tabulated in table 3. Pesticide products cause from 100 to 150 deaths a year or from 6 to 10 percent of the fatalities associated with accidental poisoning by liquid and solid substances. This is in keeping with the findings of regional studies on nonfatal poisonings that have been conducted by the American Academy of Pediatrics, the American Red Cross, and various poison control centers. The most recent reports from these groups indicate that injuries from pesticides accounted for 9.8 percent of the 3,877 reported cases of accidental poisoning recorded by the Accident Prevention Committee of the American Academy of Pediatrics,[9] for from 6 to 8 percent of the several hundred cases of poisoning reported to the Washington, D. C., chapter of the American Red Cross annually from 1952 through June, 1956, and for 6 percent of the 2,944 cases reported to the Poison Control Center of the New York City Department of Health from April through December, 1955. Attention is also directed to the rather large number of fatal accidents associated with the older pesticides. Table 3 shows the surprising constancy with which these figures recur from year to year.

Control of Poisoning from Pesticides

The control of poisoning due to pesticides is a complex problem for which there is no single or pat answer. It is generally agreed that increasing the public awareness of safety, increasing the use of precautionary labeling, and improving the accuracy of statistics on poisoning would go far in controlling the incidence of unintentional intoxication.

Increasing the public awareness of safety without frightening persons away from the use of pesticides is a ticklish problem. It is difficult to make people "poison conscious" without scaring them in the process; yet, we must advise users about the dangers in the use of pesticides if we are to improve the safety record of these agents. Constant warnings about the toxic properties of pesticides undoubtedly tend to create anxiety in some users, but this is the price we must pay to educate the public.

Precautionary labeling is an important weapon in the drive to increase the awareness of safety. Pesticides used in agriculture are subject to registration and labeling when they are sold in interstate commerce and within 39 states. Seven states have no law, and two states have regulations that pertain to one product only. In three other states, the regulations do not apply to household and industrial pesticide products.

The difficulties of obtaining information about products sold in those states with inadequate or with no regulations is illustrated by the following report recently received from a Houston, Texas, physician:

> Under separate cover, I am mailing to you an insecticide pad for your study. The history and effect of it . . . [are] interesting.
>
> Early in December this pad was bought from a door to door salesman. The company is not listed in the current phone directory. An order for more pads was filled through the telephone number listed on the pad. Since the first of January this phone number is reported as a non-operating number. The local Better Business Bureau has no informa-

TABLE 2.—*Deaths from Accidental Poisoning by Certain Solid and Liquid Substances and by Specified Gases and Vapors in United States, 1950**

Substance	Total Deaths, All Ages
Accidental poisoning by solid and liquid substances	1,584
Morphine and other opium derivatives	22
Barbituric acid and other derivatives	409
Aspirin and salicylates	90
Bromides	14
Other analgesic and soporific drugs	109
Sulfonamides	4
Strychnine	23
Belladonna, hyoscine, and atropine	2
Noxious foodstuffs	18
Alcohol	246
Petroleum products	112
Industrial solvents	34
Corrosive aromatics	78
Mercury and its compounds	23
Lead and its compounds	61
Arsenic and antimony, and their compounds	58
Fluorides	9
Other and unspecified solid and liquid substances	204
Other and unspecified drugs	59
Accidental poisoning by gases and vapors	1,769
Utility (illuminating) gas	1,012
Motor-vehicle exhaust gas	351
Carbon monoxide gas	251
Cyanide gas	9
Other specified gases and vapors	105
Unspecified gases and vapors	41

* Data adapted from Vital Statistics-Special Reports, National Summaries, Deaths and Crude Death Rates for Each Cause, by Race and Sex: United States 1950, vol. 37, no. 6, National Office of Vital Statistics, Department of Health, Education and Welfare, Feb. 16, 1953.

TABLE 3.—*Deaths from Accidental Poisoning by Pesticides in United States**

Substances on Death Certificate	1946	1947	1949	1950	1952	1953	1954
Arsenic compounds	63	48	57	58	49	52	...
Fluorides	...	...	19	9	5	6	...
Tobacco derivatives	11	10	...	...	...	...	...
Other compounds	77	59	54	53	50	52	52
Arsenic mixture	...	...	1	1	...	1	2
Cyanides	...	...	2	1	...	8	5
Chlorinated hydrocarbons†	6	5	8	4	7	8	5
Fluoride mixtures	14	10	...	...	...	...	2
Nicotine	...	...	5	5	9	3	8
Parathion, TEPP	...	...	8	9	5	5	6
Phosphorus	23	17	16	19	16	15	12
Naphthalene	...	1	...	1	...	...	1
Thallium	...	...	...	...	...	...	2
Unidentified‡	34	26	14	13	13	12	9
Total deaths from pesticides	151	117	130	120	104	110	
Total deaths from solid and liquid substances	1,536	1,504	1,634	1,584	1,532	1,391	

* Data from Moriyama.[8] Deaths assigned to categories E886, 876, 887, and 894 of sixth revision of International Lists of Diseases and Causes of Death and to 179a, e, h, and x of fifth revision.

† Benzene Hexachloride (1), chlordane (1), DDT (27), dichloroethyl ether (2), endrin (1), lindane (1), lindane and TEPP (1), toxaphene (4), and other (5).

‡ Unidentified compounds: ant poison (4), bedbug powder (1), cotton poison (2), coyote poison (1), fly spray (8), fruit spray (4), grasshopper poison (1), insect poison (13), moth spray (1), peach spray (1), rat poison (73), roach paste (7), sheep dip (1), termite poison (1), tobacco dust (1), tree killer (1), and weed killer (1).

tion on this company. The local Health Department has no information and no jurisdiction except when used in a public eating establishment. The local Pure Food and Drug Office of the Department of Health, Education and Welfare claims no jurisdiction. They also said the Department of Agriculture which does control insecticides would have no jurisdiction unless it was sold inter-state. Texas has no laws controlling sales of such products.

I am sending you one pad but retaining one other pad in reserve. All other pads in the possession of me or my friends have been destroyed. I would appreciate hearing from you as to the active agent and as to what precaution to the public might be taken.

The physician's frustrating attempt to get information on the composition of the product was prompted by a suspicion that the product was involved in a case of poisoning. Analysis of the product in question showed that it contained *p*-dichlorobenzene. After review of this report, the Committee on Pesticides recommended that copies of this report be brought to the attention of all interested health and regulatory agencies.

Comment

By giving the foregoing report, containing case reports of poisonings and the statistics on the incidence of injury, it was not my purpose to exaggerate the fear of the dangers of pesticides. Rather, I hope these facts will stimulate greater awareness of hazards, the means for recognizing intoxication, and the prevention of accidents. Certain accidents are unavoidable at our present level of social and scientific accomplishment; however, many other accidents are due to lack of information, misjudgment, and gross carelessness. While education has been advised, it has not been universally applied as a measure to overcome these malefic influences. It is through education that the control of accidents due to pesticides can be accomplished most profitably.

535 North Dearborn (10).

References

1. Conley, B. E.: "Poisoning by Insecticides," **Modern Treatment, A Guide for General Practice**, edited by A. Smith and P. L. Wermer; Paul B. Hoeber, Inc., New York, 1953.

* * * * *

8. Moriyama, I. M.: Personal communication to the author.

9. Press, E.: "Poison Control Centers: A New National Movement," *A. M. A. Arch. of Indus. Health.* 13:305-307 (March) 1956.

Phkuhli

—Moffett Chicago

Left: Ralph Kuhli.

KUHLI PROMOTED TO STAFF EXECUTIVE FOR PROGRAMS

IN SEPTEMBER 1957 Ralph Kuhli was promoted to staff executive for programs in the office of the assistant general manager of the National Safety Council, W. G. Johnson. The Home Safety Division is one of the program departments reporting to the assistant general manager of programs.

Philip Dykstra, senior consultant in the Home Safety Division, has been assigned responsibility for the Home Safety Inventory and for contacts with public health departments.

Among Mr. Kuhli's new responsibilities will be directing and assisting the managers of the program departments in developing programs and services which will attain the objectives of the several departments and thus the council's objectives. Another responsibility will be evaluating effectiveness of programs, services, and future plans; initiating planning to fill major program and service deficiencies; and ensuring that plans and programs reflect up-to-date statistical and research findings.

HOW TOXIC IS IT?

DR. GOSSELIN was at the Department of Pharmacology, School of Medicine and Dentistry, University of Rochester, Rochester, N. Y., at the time this paper was read. He is now a professor of pharmacology in the Department of Physiological Sciences at the Dartmouth Medical School, Hanover, N. H.

This paper has been condensed by the author for the *Home Safety Review* from one which was read in the Symposium on Childhood Poisons before the Joint Meeting of the Section on Pediatrics and the Section on Preventive and Industrial Medicine and Public Health at the 105th Annual Meeting of the American Medical Association, Chicago, Ill., June 12, 1956. The complete paper has been published in the *Journal of the American Medical Association*, vol. 163, No. 15, pp. 1333-1337, April 13, 1957.

In Table 2 below a number of types of household products are listed and rated by the amount that, if consumed, would cause death. Although this rating has been established mainly for the use of clinicians and doctors, these are the types of products about which parents of preschool children should exercise precaution. Perhaps a parent could inquire when purchasing a product which is the least toxic or nontoxic of those that would do the job best. Certainly products that might be poisonous—whether fatally so or not—should be stored out of young children's reach or under lock and key.

By Robert E. Gosselin, M.D., Ph.D.

ONE OF THE commonest questions of the medical practitioner consulting the toxicologist at a poison control center is "How toxic is it?" This query reflects a widely acknowledged need of physicians for guidance when confronted with a patient known to have been exposed to a potential intoxicant. In most cases of alleged poisoning seen by pediatricians in Rochester, N. Y., and presumably elsewhere, the patient develops no significant clinical illness and often no symptoms whatever. The immediate anxiety of parents, however, is understandably as intense when a toddler has ingested a harmless furniture wax as when he has swallowed a dangerous metal polish.

Without information about the composition or toxicity of commercial products, a physician is forced to examine every patient who has ingested accidentally or willfully anything that is "nonedible." Given reliable toxicity data, a pediatrician in many cases can confidently reassure an anxious mother without making an unnecessary house call to see the child. In other cases, toxicity information makes it possible for the physician to reach a prompt and intelligent decision about the need for hospitalization, diagnostic tests, and prophylactic treatment even before the patient exhibits signs and symptoms of intoxication. To the medical practitioner, therefore, toxicity data are useful chiefly as guides in formulating a realistic prognosis.

After the physician learns the brand name and the approximate amount of the product ingested, he requires next a statement of the product's composition and an estimate of its inherent toxicity. Recently Gleason, Hodge, and I[1] [see page 12] compiled data on more than 15,000 commercial products that are currently marketed for use in the home and on the farm. The original report[1] was designed as a reference book for use in emergency cases of chemical poisoning. The present report deals only with the ranges of toxicity that are encountered in common home and farm products and illustrates the use of numerical "toxicity ratings" as a convenient way of answering the question, "How toxic is it?"

TABLE 1.—*Definitions of Toxicity Ratings*

Toxicity Rating or Class	Probable LETHAL Dose (human)	
	mg./kg.	For 70 kg. man (150 lbs.)
6 Super toxic	Less than 5	A taste (less than 7 drops)
5 Extremely toxic	5-50	Between 7 drops and 1 teaspoonful
4 Very toxic	50-500	Between 1 tsp. and 1 ounce
3 Moderately toxic	500-5 gm./kg.	Between 1 oz. and 1 pint (or 1 lb.)
2 Slightly toxic	5-15 gm./kg.	Between 1 pt. and 1 quart
1 Practically non-toxic	Above 15 gm./kg.	More than 1 quart

This table is reproduced with permission from Gleason, Gosselin, and Hodge.[1]

Procedure

Two ways are available for assessing the toxicity of commercial mixtures. First, the product itself can be tested in a series of laboratory animals to obtain estimates of the mean lethal dose and to determine the nature of the toxic syndrome and the organ, if any, that is the target of damage.

A second and more generally available method requires a toxicological appraisal of each ingredient in the commercial mixture. If one knows the toxicity of each component when it is tested separately, the magnitude of the lethal dose of a commercial mixture can be inferred if one is willing to assume that all the constituents act independently and have neither additive nor antagonistic effects. Except when two or more ingredients are chemically related, this simplifying assumption is generally permissible as long as the final estimates are not intended to be precise. Another way of stating the current working hypothesis is this: The presumptive lethal dose of a commercial mixture is the small-

TABLE 2.—*Representative Commercial Products in Each Toxicity Class*

Class 1 (practically nontoxic)	Class 2 (slightly toxic)	Class 3 (moderately toxic)
Foods	Cosmetics (most)	Polishes (metal, wood, shoe, stove)
Candles	Adhesives (most)	Cosmetics (hair dyes, tonics, permanent waves; liquid lipstick; nail polish, enamel, remover; perfumes)
Mucilages and pastes	Lubricating oils	Cleaners (window, stain removers)
Cosmetics (especially baby products)	Lubricants (most)	Adhesives (rubber, linoleum, roofing, plastic cement)
Abrasives	Soap products and detergents	Bleaches
Pure soaps	Waxes (general, wood, window)	Motor fuels
Lead pencils	Polishes (porcelain, some furniture)	Lighter fluids
Modeling clays	Inks (some)	Repellents (insect, cat, dog)
	Incense	Antifreeze
		Brake fluids
		Mothballs (most)
		Preservatives (brush, canvas, roof)
		Matches
		Inks
		Agricultural chemicals (many)

Class 4 (very toxic)	Class 5 (extremely toxic)	Class 6 (super toxic)
Disinfectants (most, such as those for garbage cans)	Drain and sewer cleaners (caustics)	Insecticides, Fungicides, Rodenticides, Herbicides (a few)
Dry cleaner solvents (some)	Fireplace flame colors (blues and greens)	
Degreasers (metal, etc.)	Insecticides and fungicides (some)	
Depilatories (some)	Rodenticides (some)	
Drain cleaners (some)	Herbicides (some)	
Naphthalene moth repellents		
Rust removers		
Radiator cleaners		
Leather dyes		
Indelible inks		
Fire extinguisher liquid		
Agricultural chemicals (many)		

est quantity that contains a fatal amount of any one of its constituents. By the use of this operational definition, the toxicity of a mixture is ascribed solely to the action of one of its ingredients or of one group of chemically related ingredients, all others being regarded as diluents.

To locate toxicity data about all ingredients, many different sources and kinds of information were used. Spector[2] has recently summarized the published values of the mean lethal dose (LD_{50}) in laboratory animals for a large number of substances. Toxicity reports that were furnished by manufacturers proved to be invaluable sources of unpublished data about many newer chemicals.

Results

Table 1 defines the concept of numerical toxicity ratings, or classes of toxicity, by outlining the criteria used in assigning them. This scheme of classification, with its arbitrarily established limits, has been modified from a table originally designed by Hodge and Sterner[3] for industrial chemicals, not consumer goods. It is evident that every chemical substance and every mixture of substances must belong in one of these six toxicity classes.

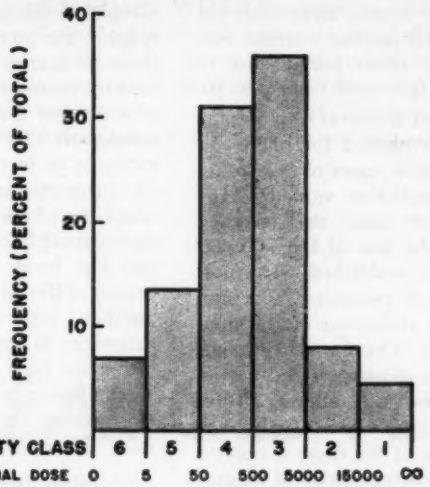
In surveying the products on the consumer market, it becomes apparent that, irrespective of the brand name, the majority of items intended for the same use warrant the same toxicity rating. Of course, there are exceptions in which specific brands deserve a lower rating and others a higher rating than the typical example. Table 2 indicates the kinds of products that are commonly found in each of the six toxicity classes. Admittedly, too few examples of some commercial products have been studied to justify the claim that the most representative toxicity rating was selected in every instance. Even if a more complete survey demonstrates that some of these ratings should be revised, the list illustrates convincingly that the greatest diversity of products is found in toxicity class 3. Comparatively few kinds of consumer goods warrant a rating of 5 (extremely toxic) or 6 (super toxic); these include some of the concentrated caustic cleaners and a minority of the insecticides, fungicides, herbicides, and rodenticides. The largest variety of cosmetics and toiletries is found in class 2 (slightly toxic), but most of the perfumes and many of the hair and nail preparations are placed in class 3, because of their high concentrations of alcohol and other organic solvents.

If one chooses a broad category of products, examples of these products can usually be found in several toxicity classes. Table 3 indicates the relative frequency with which the various toxicity ratings are encountered in diverse types of commercial products. Toxicity class 3 is seen to contain the largest number of items in all but one category of table 3.

If one has no information about a particular product except that it is nonedible, there appears to be an

TABLE 3.—*Frequency Distribution of Toxicity Ratings of Products in Various Categories*

Type of Product	No. of Rated Products	Products Not Rated, %	% of Products with Toxicity Ratings of 1	2	3	4	5	6
Cosmetics (toiletries)	365	...	5.8	45.2	45.8	2.2	1.1	...
Drugs (human)	1,100	4.9	2.0	32.2	54.4	10.5	0.9	...
Drugs (veterinary)	237	4.8	1.7	18.6	49.8	19.8	9.7	0.4
Cleaners (polishes, waxes)	684	...	2.3	14.9	53.7	28.5	0.6	...
Paints and accessories	736	1.6	0.3	5.8	57.6	36.0	0.4	...
Insecticides, miticides, and so forth	1,344	3.6	0.1	9.2	47.1	30.3	8.6	4.8
Agricultural fungicides	189	2.1	...	12	51	24	12	1
Herbicides (weed-killers, defoliants)	148	1.3	...	3	63	26	5	3
Rodenticides	111	9.0	1	46	23	16	6	7
All agricultural products including miscellaneous	1,929	4.0	0.2	11.4	47.0	29.4	8.0	4.0
All nonagricultural..	3,295	2.2	2.2	23.0	54.1	19.4	1.3	...
Total of all types....	5,224	2.9	1.5	18.8	51.6	23.1	3.5	1.5



Distribution of toxicity ratings of 1,002 ingredients found in commercial products. Lethal doses are given in milligrams per kilogram of body weight.

even chance that it has a toxicity rating of 3. In table 3, only the higher and lower classes reveal distinct differences between various kinds of commodities. For example, the half of the number of cosmetic products that lies outside of class 3 is found almost entirely in lower (less toxic) classes. In contrast, most agricultural chemicals that do not belong in class 3 warrant a higher (more toxic) rating. The rodenticides are an exception, because two of the ingredients that occur most frequently are warfarin and alpha-naphthylthiourea (ANTU), both of which have comparatively low single-dose toxicities in man.

Mixtures with toxicity ratings of 5 or 6 are always in a minority. Among insecticides and pesticides generally, these two ratings appear in products that contain high concentrations of nicotine, its salts and derivatives; cyanides; fluorides; arsenic; various organic phosphate compounds, such as parathion, tetraethylpyrophosphate (TEPP), sulfotepp, demeton (Systox), octamethylpyrophosphoramide (OMPA), ethyl *p*-nitrophenylthionobenzenephosphate (EPN), and para-oxon; the more toxic cyclodienes, such as aldrin, dieldrin, endrin, and perhaps isodrin; and supposedly various carbamates such as Pyrolan and Isolan (no examples in this compilation).

The very toxic agricultural fungicides are usually those that contain high concentrations of arsenic or mercury (both organic and inorganic forms), sometimes cadmium salts, and perhaps rarely actidione (Cycloheximide). A toxicity rating of 5 or 6 for a herbicide is generally due to its high content of arsenic or of one of several dinitrophenol derivatives; rarely disodium Endothal may be responsible, but no product with a concentration above 19.2 percent was located. Perhaps phenylmercuric compounds may be responsible, but none appeared with more than 10 percent concentration. Rodenticides warrant toxicity ratings of 5 or 6 when they contain high concentrations of strychnine, arsenic, fluorides, fluorosilicates, and barium salts; perhaps yellow phosphorus and thallium sulfate should be included, but no product with more than 3 percent of phosphorus or 5 percent of thallium was encountered.

In comparing table 3 with the figure, it can be seen that the toxicity ratings of products cluster around the center more than do the ratings of ingredients. This must mean that, of those constituents that commonly determine the toxicity rating, highly toxic ingredients are generally marketed in a dilute form, while ingredients of low toxicity are often found in high concentrations.

Comment

Numerical toxicity ratings were proposed originally for the use of industrial toxicologists to fill "a long-felt need for a simple, understandable expression of the degree of toxicity."[3] Physicians and clinical toxicologists also need a simple scale of reference for describing and comparing grades of toxicity. A numerical scale, with a parallel set of descriptive phrases, is believed to meet this requirement, and specifically the six ratings or classes defined in table 1 appear to be appropriate for the needs of physicians. As illustrated here, these ratings aid in compiling, analyzing, and presenting data about the degrees of toxicity encountered in household chemicals, including commercially available mixtures. The ratings provide a simple and unambiguous way for toxicologists to communicate data on toxicity to clinicians.

Limitations of Ratings.—To use toxicity ratings effectively, however, the following several implications and limitations must be appreciated:

1. The rating is based on mortality, not morbidity; it is really a rating of lethality. In general a clinically significant illness may be expected to occur after the ingestion of a dose as small as one-tenth the lethal dose.

2. The rating is based on the acute toxicity of a single dose when taken by mouth or by gavage. Other regimens and other routes of administration are not represented, and the rating tells nothing about the toxic hazard, if any, when a product is used in the proper or customary way.

3. The toxicity rating reflects an estimate of the probable or mean lethal dose, not the minimal fatal dose. Perhaps because of personal idiosyncrasy, hypersensitivity, or predisposing disease in the patient, most lethal doses recorded in the clinical literature are considerably lower than those implied by the toxicity ratings that were assigned here.[4]

4. The definition of toxicity ratings implies that lethal doses are proportional to body weight. In table 1, common units of measure are used to describe the approximate

lethal ranges for an adult person of average size (body weight of 70 kg. or 150 lb.). For patients who are heavier or lighter, probable lethal doses vary proportionately, and they can be estimated readily from the values of milligrams per kilogram of body weight given in table 1. The parameter of age is ignored because there is no conclusive evidence that the influence of age on toxicity can be generalized or that it is a major factor in the action of most poisons.

5. The clinical data on only a few substances are adequate to establish a toxicity rating. Most values were founded upon laboratory determinations of the mean lethal dose (LD_{50}) in small laboratory mammals (rats, mice, guinea pigs, rabbits, and sometimes cats, dogs, and monkeys). Whenever available, however, clinical data and even clinical impressions have been given precedence. Clearly all the ratings are tentative; if necessary, any rating can be revised at any time within the framework of table 1.

6. For most corrosive agents, such as mineral acids, alkalies, and bleaches, a toxicity rating has limited value. In cases of poisoning due to these agents, death is usually the result of a severe injury to local tissue, with secondary complications, such as toxemia, shock, perforation, hemorrhage, infection, and obstruction. The intensity of the local lesion and of its sequelae is determined often by the concentration of the corrosive substance, while the volume and dose are secondary considerations. For such agents, no single toxicity rating is an appropriate measure of lethality, unless the concentration is also specified. No simple parameter describes this relation in a way that is clinically useful.

7. The criticism that the toxicity rating does not define the lethal dose with enough precision to make it clinically useful is easily answered. As long as products available on the consumer market vary more than one hundred thousandfold in lethality, estimates with even a tenfold range of uncertainty are worthwhile. Whatever the scheme of classification, no rating can be more accurate than the clinical and laboratory data on which it is based. Even with old and established poisons, current estimates of the mean lethal dose in man vary by factors of from 2 to 10.

8. Precision is unnecessary insofar as a rating is needed for making a prognosis in most cases of poisoning. Thus the predictive value of any rating depends upon the accuracy with which the size of the ingested dose can be established. In most clinical cases of poisoning, a precise and accurate statement of the dose is unavailable. On the other hand, whenever the identity of the poison is known, one can almost always make some reasonable inference about the size of the dose, if only to estimate the correct order of magnitude. For most purposes, the toxicity rating does not need to be more precise.

9. -The current analysis does not tell what type of product is responsible for the largest share of cases of acute poisoning in the home or on the farm. To make any predictions about the incidence of poisoning, elaborate information is necessary first about the availability of various poisons. Perhaps this can be measured crudely by the volume of sales, in which case the number of retail units is probably more pertinent than the total poundage. Other factors also influence the extent to which a substance is a toxic hazard. For example, odor, taste, color, volatility, stability, packaging and labeling, size of the retail unit, and conventional techniques of storage by the consumer—all contribute to the epidemiology of chemical poisoning. Inherent toxicity of an agent is only one and often a minor factor in its hazardousness, but it is the only element in the list that concerns the physician who must treat a patient known to have swallowed a potential intoxicant.

Summary and Conclusions

A numerical system of toxicity ratings, modified from a scheme used by industrial toxicologists, is a potentially useful way for physicians to describe and compare degrees of acute toxicity of common household poisons. Over 15,000 specific products on the consumer market have been assigned toxicity ratings.[1] A sample of this compilation was analyzed in the present study in terms of these six ratings to show the range of toxicity encountered in various types of consumer goods. In clinical situations, the chief value of a toxicity rating is in formulating an immediate prognosis in cases of poisoning when the physician knows what and approximately how much of the poison has been ingested. Because brands differ significantly from one another, especially within certain categories of merchandise, a specific rating for each product is desirable. These ratings are clinically serviceable indexes for answering the question, "How toxic is it?"

43 College Street, Hanover, N. H.

References

1. Gleason, M. N.; Gosselin, R. E.; and Hodge, H. C.: *Clinical Toxicology of Commercial Products, Acute Poisoning (Home & Farm)*, p. 18, the Williams & Wilkins Co., Baltimore, 1957.

2. Spector, W. S.: *Handbook of Toxicology*, vol. 1, W. B. Saunders Company, Philadelphia and London, 1956.

3. Hodge, H. C., and Sterner, J. H.: "Tabulation of Toxicity Classes," committee report, *Am. Ind. Hyg. Association Quart.* 10:4 (Dec.) 1949.

Set of Books

Available from the University of Michigan Publications Distribution Service

412 Maynard Street
Ann Arbor, Mich.

Home Accident Prevention, set of 5 books..... $6.00

Unit Prices:

Home Accident Prevention proceedings—Jan. 20-22, 1953 ..... $1.00

Home Injuries..... $1.50

Appendix A to Home Injuries $1.50

Appendix B to Home Injuries $1.00

Home Accident Prevention proceedings—June 19-21, 1956: the *Second Conference on Prevention of Home Accidents* $1.00

THIS SCRIPT was produced by the WGAR Broadcasting Company and the Ohio State Safety Council, Cleveland, Ohio. H. G. J. Hays, executive secretary and treasurer, Ohio State Safety Council, states that this is one of a series of scripts the anecdotes for which were derived from a number of sources, among them newspapers and the annual Freak Squeak story of the National Safety Council. Occasionally someone sends an anecdote to him.

A 3-YEAR-OLD PLAYS NURSE

VOICE: "So Help Me; It's the Truth!"

ANNOUNCER: Station ——— and the Ohio State Safety Council present "So Help Me . . ." a 5-minute program featuring the Voice of Safety as he tells of some of the freak squeaks, the crazy goings-on as your fellow citizens got themselves involved in accidents. This evening, for instance, the Voice of Safety tells us about a 3-year-old who played nurse.

VOICE: "So Help Me; It's the Truth!"

It happened in California where many extraordinary events are said to take place. Trying to take her own temperature, the little girl used a weather thermometer. She bit off the bulb and swallowed some of the fluid. Her condition, after sudden squalls and use of the stomach pump, was reported as fair, but unsettled, with rising mercury.

Believe it or not, this type of accident is pretty common because many commonplace things can be dangerous to children: mother's scissors, dad's knife or other tools, medicines that are brightly colored—even some of the household cleansers that are used every day. We don't realize that children do most of their learning by imitating what they see other people doing. The 3-year-old in Pasadena was not unusual. Thousands of youngsters play nurse or doctor every day. That puts the burden of safe instruction squarely on the shoulders of the adults who are around the child regularly.

Did you ever notice that almost every day's paper tells of at least one child taken to the hospital because he swallowed some harmful object? Did you ever wonder how such a thing could possibly happen?

Mother left several glistening safety pins on a tray in the sewing room; sister never puts her bobby-pins or earrings out of sight. Dad leaves a broken razor blade on the bathroom window sill. The scouring powder and other cleansing aids stand on a low shelf in the closet or beneath the kitchen sink.

The medicine cabinet is unlocked, and the door opens easily. Of course, it's out of reach—until a venturesome, curious child pushes a stool over to it or climbs into the washbowl to reach it. Within that medicine cabinet, maybe, are any number of items which can be harmful to a child's digestive system if he swallows some of them, and he probably will if he's gotten so far as to open the chest door. He's just imitating someone—or he's just curious; maybe he thinks the pills are candy. A child must be nearly 6 years old before he gets out of the habit of putting things in his mouth. So what's the answer? Lock up harmful drugs, kerosene, turpentine, bleaching solution, lye, sal soda, and antiseptics. Don't rely on a high shelf or a door that's difficult to open. Some youngsters will find a way to reach or open it. Keep matches out of reach. Match heads are not the best eating in the world; besides he might try to light some and start a blaze where it's not wanted.

But children are not the only ones who need watching when it comes to drugs and medicines. Always look at the label before taking any medicine. Don't rely on knowing where the bottle is. Someone may have changed things around. A man took liniment for cough syrup and an elderly grandpa drank what he thought was a cup of black coffee; the latter turned out to be some varnish stain that his daughter was going to apply to a piece of furniture. Children have drunk kerosene and cleaning fluids thinking they were water. Never put such liquids in a cup or drinking glass; it makes them even easier to mistake for a drinkable liquid. And another thing, kids are always reaching for things which they tip over on themselves. Keep the handles of pots and pans turned so that they don't stick over the edge of the stove or sink.

Be sure that valves on your gas stove are kept tight so a child cannot open them. And a good general rule to follow is: Don't let a child imitate adults too much. He'll try some things at the wrong time with saddening results. It may be cute for him to light daddy's cigarette now, but later, when no one's *near,* his play can cost your home—maybe his life.

ANNOUNCER: You have been listening to "So Help Me . . . " presented by ——— each ——— at this time for the Ohio State Safety Council. The Voice of Safety will be back again next week with the story of another freak accident and some tips on how you and I can stay alive and healthy. "So Help Me; It's the Truth!"

THIS PAPER was read in the Symposium on Childhood Poisons before the Joint Meeting of the Section on Pediatrics and the Section on Preventive and Industrial Medicine and Public Health at the 105th Annual Meeting of the American Medical Association, Chicago, Ill., June 12, 1956. It is reprinted from the *Journal of the American Medical Association*, vol. 163, No. 15, pp. 1344-1348, April 13, 1957.

Manufacturer Looks at

By John H. Foulger, M.D.
Wilmington, Del.

EACH YEAR AT about this time the National Safety Council publishes a little book entitled *Accident Facts*. It gives the number of fatal accidents in the United States in the previous calendar year. It lists them by place (industry, farm, or home), by cause, and sometimes by the age of the victim.

Each year for several years, under the title How People Were Killed in Home Accidents, there have appeared two figures of interest to us today. In magnitude they vary little from year to year. For 1953 they are total fatal home accidents, 29,000, and deaths from solid or liquid poisons (including medicaments), 1,200. The ages of the victims who swallowed solid or liquid poisons, including medicaments, are usually charted. Among children under 5 years of age, the number of deaths for several years has been approximately 400. Of this number, some 20 to 25 percent of the deaths have been due to the swallowing of medicaments.

PROGRAMS OF VARIOUS GROUPS

This continued pattern of deaths of young children, with its implication of the occurrence of many unreported, nonfatal, but nevertheless serious cases, has in recent years attracted much attention from pediatricians. In 1950, the American Academy of Pediatrics appointed a committee on accident prevention. From its inception, the committee has been very active. Its work has led to a rapidly increasing number of poison control centers and to professional and public discussions of the problem.

Manufacturers of chemicals have been cognizant for many years of this annual toll of 1,200 lives in the home and have given much thought to means of prevention. A program of prevention must involve two phases. The first is prevention of incidents of misuse, whether accidental or intentional. The second is prevention of the serious outcome of such incidents. The first phase must involve, in part, the activity of the manufacturer and marketer of household chemicals. The second, the prevention of serious outcome, must be primarily the responsibility of the medical profession. In this phase, however, clinicians can benefit from advice from physicians who work in or with the chemical industry and who have special knowledge of the pathological changes produced by chemicals.

In 1944, the Manufacturing Chemists Association, representing manufacturers of some 90 percent of the chemicals produced in the United States, appointed a permanent committee on labels and precautionary information. This committee has formulated basic principles of labeling, which are now relied upon by a number of states (California, Illinois, Oregon, New York, and New Jersey) and by the Territory of Hawaii and the City of New York, in setting up regulations on labeling. These basic principles have been accepted by the International Association of Governmental Labor Officials. In recent years, a committee that is almost parallel in function has been formed by the Chemical Specialties Manufacturers Association. This organization is particularly interested in the marketing of small household packages.

Over many years, the larger chemical manufacturers on an informal basis have followed a procedure quite similar to that of the new poison control centers. Thus, for some 20 years, on the behalf of one manufacturer, I have personally given advice by telephone to any physician in the United States who has asked for help in handling a case of chemical poisoning. Any physician can call me day or night, whether I am in my home or in my office.

Other industrial organizations are always ready, when opportunity is given them, to give advice and to investigate cases of poisoning. Among

Child Poisoning

THE LABELING of chemicals has come to embody many principles of safety, but it cannot be the sole means of protection for children. Labels are for those who can read. Reasonable behavior in parents and all other adults is the only sound method of reducing the number of accidents in which children swallow household chemicals or drugs. Information about active ingredients can be obtained by telephone from manufacturers or from poison control centers. Treatment can be specific if the poison is known to be an acid or alkali or a nerve poison of the organic phosphate type. In most cases the prompt induction of vomiting by putting a finger down the throat or drinking warm salt water will remove much of the offending material from the child's gastrointestinal tract.

these, the National Paint, Varnish, and Lacquer Association is especially concerned with injury caused by use of their products, including those containing lead.

Reasons for Concern of Manufacturers

It is clear that the chemical industry cannot be indifferent to the problem of poisoning. Under common law, he who makes, sells, or distributes an article that is inherently dangerous to health assumes a duty to the user and to those who may be expected to participate in or be in the vicinity of the use of the article. This duty is to warn the user of the hazards to health involved in the proper use of the product. Failure in this duty makes the manufacturer, vendor, or distributor liable for monetary damages. In these days, such damages may be large amounts of money, but the manufacturer's concern over injuries by its products is not solely due to financial self-interest. The modern manufacturer realizes that, as Governor Knight of California so recently said, "financial losses can be recovered—lives never." In these days of a highly industrialized country, I, acting as a manufacturer, may make items that you, as a citizen, use; however, as a citizen, I may also be the user of your products. If I subscribe to an attitude of indifference toward injury caused by my products or your products, I or my family may be the victims. My child or grandchild could well be one of the 400 young children appearing as a statistic in *Accident Facts*. From many years of association as a physician with committees from the chemical industry, I have learned that the chemical manufacturer is acutely aware of the need for concern.

Prevention of Injury by Household Chemicals

Problems in Prohibition of Harmful Chemicals.—Several suggestions have been made for the prevention of injury caused by household chemicals. Extremists demand that harmful chemicals should never be sold for household use. They advocate absolute prohibition. This attitude, of course, is impractical. We are not all children under 5 years of age. Adults demand new tools that will lighten household chores and make life in general more attractive. What they demand, they get. The manufacturer of a particularly hazardous chemical may refuse to market it for use in the home. He may strongly advise against its use in the home. Yet, if the householder demands it, it will enter the home, for no manufacturer can police the whole country. Without his knowledge, the product that he has proscribed for household use can be bought surreptitiously from jobbers and packaged by others for home use. Too often, the individual packager and marketer of the compound is not sufficiently experienced to realize the harm he may do.

The tragic episode several years ago caused by the use of a diethylene glycol solution of sulfanilamide stemmed from the initial refusal by a large manufacturing company to sell the solution for pharmaceutical use. The chemist who wanted to use it bought it from a jobber. The jobber had received it for quite different, industrial applications.

During the last two or three years, I have personally spent much time and effort in trying to persuade surgeons not to use new, ill-defined and untried plastic materials as prostheses in humans without first making a long-term test in animals to determine suitability and safety. In this, I have so far had very little success. If the public, or any section of the public, demands a particular product for its use, it sooner or later gets it, regardless of the wishes and plans of the initial manufacturer.

Problems in Labeling.—Labeling of household packages of chemicals has been advocated as an infallible solution to the problem. From some quarters arises the cry, "Give us a

labeling law." Labeling has been carefully studied by chemical manufacturers for many years. While the principles of labeling of the Manufacturing Chemists Association committee are primarily intended for use by makers of industrial containers of chemicals, the problem of household packages has not lacked consideration.

The M. C. A. labeling principles require that a label on an industrial container carry the following items: (1) the name of the product, preferably the chemical name; (2) a single word—"danger!" "warning!" or "caution!"—that expresses the relative severity of the hazard that the product affords; (3) a statement of the type of hazard, such as "causes burns," "rapidly absorbed through the skin," or "vapor extremely hazardous"; (4) precautionary warnings, such as "avoid contact with the skin or eyes" or "keep away from heat or open flame." In the words of a federal statute concerning the labeling of insecticides, the label should constitute "a warning or caution that may be necessary and if complied with adequate to prevent injury to living man." We consider the words "if complied with" to be key words. When an industrial product is extremely harmful, the label may carry the word "poison," perhaps a skull and crossbones, and, in addition, a recommended simple first-aid treatment.

The immediate application of these principles of labeling to household packages of chemicals is not as simple as it may appear, especially when we remember that we are concerned with the protection of young children. Industrial containers of chemicals are large and offer large surfaces for labeling. Household containers are small, with little available surface for labeling.

Containers of industrial chemicals need not carry directions for use. The company buying the chemical is presumed to know how it wants to use it. It can obtain additional technical assistance from the original manufacturer in the form of bulletins or by word of mouth or letter. The household container must carry directions for use, sometimes rather detailed directions. The householder is not presumed to be skilled in the use of chemicals, and improper use may cause damage to property as well as to health. Thus, little space may be left on the label for precautionary statements. Yet, to be effective, such statements must be legible and in such contrast with the other words on the label that they at once attract attention. A label that can only be read with a magnifying glass is ineffective.

In industry, chemicals may injure by passage through the skin, by contact with the skin, or by passage through the respiratory tract. Except in horseplay they are seldom swallowed. Thus, the hazards of skin contact, penetration, or inhalation alone need be considered in the labeling. Injuries to children from household chemicals are almost entirely due to ingestion. To add such phrases as "harmful if swallowed" or "may be fatal if swallowed" to an already congested label space increases the problem of available space. One cannot omit all reference to other hazardous physiological routes, for adults must also be protected. We cannot design the label for the benefit of children alone.

It is a basic principle that labels should indicate the degree of hazard associated with proper use of a product. Apart from medicolegal implications of labels bearing too little or too much information, it would be extremely costly in industry to suggest for the use of harmless, common salt the same precautions, special handling equipment, special ventilation, and personal protective devices for workers that are necessary for safe use of dangerous chemical products such as tetraethyl lead. In the home, and especially when young children have to be considered, the problem is different. The home is not and cannot be equipped from the point of view of safety in the same way as is an industrial plant. To a young child anything other than water, milk (usually), or the child's own proper diet may be harmful or even fatal if swallowed. To a child, there may be only one degree of hazard.

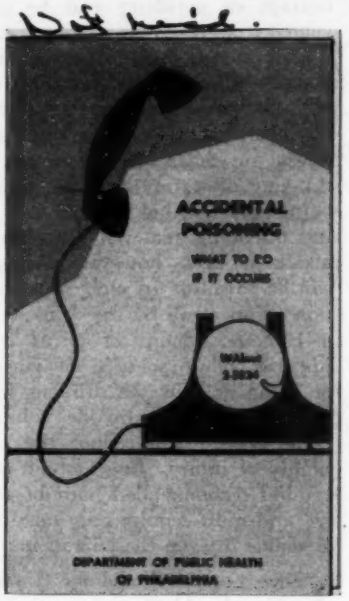
Should manufacturers, therefore, discard the principles of good labeling and mark every chemical for home use as "harmful if swallowed" or "may be fatal if swallowed," or, as some demand, should manufacturers label all household packages solely with the word "poison"? If anything that the young child swallows other than water, milk, or his own proper diet could injure him, then logically we should label as hazardous mustard, table salt, pepper, vanilla extract, and in fact almost everything chewable or ingestible in the home. This is obviously absurd. Further, the medicolegal problems involved in overemphasis or underemphasis of the degree of hazard attending the proper use of products hold for products used in the household as much as for those used in industry, and they cannot be neglected. It can be seen easily that the problem of labeling is not a simple one.

The causes of death in the home as listed by the National Safety Council show that for children of under 5 years of age the causes of death in order of decreasing magnitude are mechanical suffocation, 1,420; fires, 1,210; chemicals (solids or liquids), 410; falls, 380; firearms, 100; poisonous gases, 50; and others not specified, 2,500. (The figures, given in round numbers, are averages for the years from 1951 through 1954). We do not label everything that might cause a fall or the mechanical suffocation of a child. We do not label firearms or matches.

For whom is the label intended? Obviously not for the child. If intended for the parent, it will be effective only if read and its warning heeded. Is the warning actually heeded?

REPORT OF RECENT ACCIDENTS: Here are examples of recent accidents that were reported to me. A young mother was painting her kitchen. Her boy, about three years of age, was playing nearby. She was called to the telephone. When she came back into the kitchen, the child had one foot in the paint can. She removed the boy's foot from the paint can and began to remove the paint from the boy by using turpentine. She was again called to the telephone. When she returned,

Philadelphia Health Department Publishes Leaflet



A LIMITED RUN, 25,000 copies, of a leaflet *Accidental Poisoning; What to Do If It Occurs* (left) was published by the Department of Public Health, Philadelphia, Pa., in order to test it, both in appeal and in appearance. As a result, the Department planned to revise the pamphlet into a six-panel leaflet with more illustrations and a generally more open appearance and expected to have another 100,000 copies printed.

Emil T. Tiboni, chief, Housing Hygiene and Accident Prevention Section, Division of Air Pollution Control and Environmental Sanitation of the department, has described their plans for distribution of the pamphlet as follows last May:

"As to distribution, we intend to try to saturate the community. In addition to our usual health education channels, we have two major channels in mind. . . ." They have been negotiating one channel which would involve the placement of a rack in each of the approximately 1,200 retail drugstores in the city for the distribution of this pamphlet.

"The other major channel," he stated, "would be through the school systems. We have already obtained a tentative agreement from the public school system which might distribute 100,000 copies or so and we expect to approach the Catholic school system also. The latter system contains about 35 per cent of the school population of the city."

He also stated that the pamphlet will be available in both English and Spanish.

the child was drinking turpentine. Would any label have prevented this incident? Suppose the mother had carefully placed the paint can or the turpentine can on a shelf that she thought would be out of the reach of the child. Could anyone predict that the boy, attracted by either of these cans, would not have climbed on a box, chair, or table to reach the shelf and would not have fallen and broken an arm, leg, or his neck?

In a western state another accident occurred. Three children swallowed a solution of a weed killer. It happened in this manner. The father had made up a solution to be used in the yard. He put the portion left over in an ordinary gallon bottle. The solution was yellow-colored. The mother, thinking it was fruit juice, put it on a cellar shelf with other bottles of fruit juice. On a hot day the children were thirsty. She gave each a glass of the yellow-colored solution of weed killer to drink. Luckily, it was relatively harmless, and no injury resulted. The original package of chemical weed killer was properly labeled according to federal and state laws.

A third incident does not involve a child, but it illustrates the attitude of adults toward labeling. A worker, addicted to drinking wine, decided to take a last drink before going on the night shift at his plant. He went to a cupboard, took down a wine bottle containing a reddish liquid, poured out a glassful, and drank it. About 2 a.m., he suffered severe abdominal pain and became dizzy and nauseated. He decided to go home but collapsed before he could get out of the plant. While still conscious, he was able to tell his fellow workers that he had drunk not wine but antifreeze containing ethylene glycol, which had recently been drained from the radiator of his automobile and stored in a wine bottle in the wine cupboard. He had left the original wine label intact. The liquid looked like some of his wine.

Labeling could not have prevented any of these incidents. Indeed, it could not conceivably prevent some 20 to 25 percent of the deaths of young children reported yearly as due to medicaments. Barbituric acid and its derivatives and, recently, aspirin and the salicylates are the major single causes of deaths of young children in the home. Drugs are labeled in the original containers received by the pharmacist, but they seldom enter the patient's home in labeled boxes or bottles. They usually carry only the physician's directions, such as "Take two pills before meals." When the therapeutic need for a drug is ended, the drug may be set aside. A few weeks later no one in the household, not even the patient, may remember that it is in the house. Sometimes no one remembers what the drug really was. The patient may never have known. It would be a very difficult matter to remedy such situations by means of a labeling program.

PROBLEM OF PROPER NAMING: The proponents of labeling as a cure for our problem demand that the name of the hazardous chemical be on the label. We consider this to be good practice and essential in industrial labeling, but would it be as effective in labeling a household product? The argument in favor of this practice is that, with the name of the product on the label, the at-

tending physician will at once know how to treat his patient. If I were to tell a physician, however, that I had swallowed *p*-aminobenzoyl diethylaminoethanol, or (2,4-dichlorphenoxy) acetic acid, or ortho-ortho-diethyl-ortho-*p*-nitrophenyl thiophosphate, how many physicians would know what these compounds were, what their toxicological actions are, and how to treat me? The first, as a matter of fact, is procaine. The second is a relatively harmless weed killer, usually termed 2,4-D. The third is a deadly nerve poison, an insecticide called parathion. These three compounds have common names, but a majority of the new chemicals manufactured every year and perhaps introduced into the household may not yet have a common name.

No scheme of labeling, therefore, can be considered as a sole, satisfactory means of preventing incidents leading to injury of young children. Labeling of all materials with the same words ultimately leads to the disregard of all labels. This would be a very dangerous situation for adults as well as for children. The supplying of a proper label is only a step in a preventive program, and this step must be used with reason and care. Labeling must be supplemented by education and by the use of common sense. Chemicals are tools. Any tool used with care and good sense can offer great benefits. Any tool used with carelessness can create great harm.

Need for Care and Good Sense.—If one carefully studies the causes of fatal accidents in the home, in industry, and on the highways, one must admit that by far the greater proportion is due fundamentally to lack of care and good sense. We cannot expect the child to use care or to have the required degree of knowledge. The parents must employ care and good sense on behalf of the children.

It is noticeable that, among the causes of fatal injuries to children under 5 years of age listed in *Accident Facts*, sharp-edged or pointed tools, such as knives, forks, or ice-picks, do not appear. They are in the home. Presumably, therefore, the parents keep them away from children or children away from them. This, no doubt, is the result of experience. It should not be too great a burden to keep all medicaments and all chemical household tools away from children. None is harmless to a child regardless of whether it is labeled. All products should be treated with the same respect in the home as are sharp-edged tools.

Comment.—The manufacturers and marketers of chemicals are continuing their efforts to find a good scheme of labeling. Good labeling practices will not necessarily result from legislation. A label is useful only so long as it is read and its information is followed. The removal of labels from original containers and the storing of contents of originally labeled containers in other containers that are not labeled are common practices in the home. These are important causes of injury caused by household chemicals. They are definitely a major factor in deaths of young children.

Since carelessness and lack of common sense are involved in a majority of the accidents occurring in the United States each year, the inculcation of principles of reasonable and sensible behavior in parents and, indeed, in all adults is the only sound method of reducing the number of incidents in which children swallow household chemicals or drugs. The use of proper labeling must not be neglected, but its efficiency will definitely depend upon the reasonable and sensible behavior of parents.

Prevention of Serious Outcome

I have stated that the second phase of our total program of prevention—the prevention of serious outcome of incidents in which children swallow household chemicals and drugs—is primarily the province of the clinician. I have also stated that he may, perhaps, be assisted by physicians in industry who have special knowledge of the pathological changes produced by chemicals.

The establishment of poison control centers is a great step in the direction of the prevention of serious injury. They can be a very powerful means of education for both parents and physicians. A high percentage of accidents can be prevented from reaching a serious result if parents are taught the vital importance of promptly removing the offending material from the child's digestive tract. In most cases, the finger put down the throat or the drinking of warm salt-water should be adequate to induce vomiting. If the offending material is a strong alkali or acid, however, vomiting can be harmful. In such cases, dilution of an alkali by the drinking of a copious amount of citrus fruit juice or dilute solution of vinegar or of an acid by the drinking of a dilute calcium hydroxide solution (lime water) can reduce the possibility of injury. Any first-aid procedures recommended must be simple and must require only materials available in even the poorest homes.

The mother should be warned that she should start these first-aid procedures as soon as she discovers that her child has swallowed the drug or chemical. Let someone else call the physician. The most efficient first aid can be rendered within the first few minutes after the swallowing has occurred.

Subsequent medical treatment, of course, is also important. I would like to issue a caution. In the general field of clinical medicine, the old concept of a specific clinicopathological pattern produced by a specific etiological agent has tended to move into the background since the sulfonamides and antibiotics have allowed the control of many acute infections. We do not know the causes of many of our major chronic diseases, if for any a single cause exists. However, it is unfortunate that physicians still hold to the old concept when we talk of injuries caused by chemicals. Much valuable time may be wasted by the physician in asking "What chemical product did the child swallow?" Even if that information is immediately available, of what use is it? Chemical technology is proceeding so fast that in many cases the composition of new household tools may change frequently. Next week, for example, a paint called Yellow A

may not contain the same solvents as the paint with the same name contains this week. When the chemical is a new one, the ultimate pattern of injury that it may produce may not have been described satisfactorily even as it occurs in experimental animals. This is not due to indifference of the manufacturer but to the limited number of laboratories and personnel qualified to carry out the testing.

In the early stages, injury caused by swallowed materials may show no specific pattern but rather the general pattern of severe stress, approaching shock. The action of the respiratory and circulatory systems must be conserved. The liver and the kidneys are most apt to be influenced either indirectly by disturbance of their circulation or directly by action of the swallowed foreign chemical on the functioning cells. Vomiting may change the acid-base balance. Renal failure may change fluid and electrolyte balance. By anticipating the possibility of changes in these symptoms and organs, by giving oxygen to combat even incipient hypoxia, by attending carefully to the acid-base balance of the blood, by conserving liver and kidney function, and by carefully watching and treating abnormalities of fluid and electrolyte balance, the clinician can save many lives even though knowledge of the exact agent that caused the injury is not available to him.

Early treatment should be based on sound physiological principles. The physician should exercise restraint in the use of new drugs and particularly in the use of transfusions, unless hemorrhages or acute anemia indicate them. There will, of course, be exceptions to this general pattern. Such chemicals as the phosphate "nerve poisons" require prompt, specific therapy, but one should not stretch the concept of specificity too far lest, in place of recording a life saved, a doctor can merely record an interesting autopsy.

SUMMARY

Reduction in the number of serious or fatal poisonings from chemicals in the home involves two phases —diminution in the incidence of such cases and reduction in the seriousness of outcome. Proper labeling, for example, based on principles set forth in "Warning Labels Manual L-1," published by the Manufacturing Chemists Association, would help reduce the number of cases of injury from chemicals used in the household. It would not reduce the increasing number of cases due to drugs. The major decrease in the total number of cases can come only from education of the public to handle chemicals with the same care as they would use in handling loaded firearms or sharp-edged tools and to keep young children away from household chemicals. The seriousness of outcome of home poisoning cases can be reduced by early use of simple methods of inducing emesis. It might be diminished also by better instruction of physicians in the problems of toxicology.

1001 Orange Street (98).

Study of Chemical Labeling Laws

ONLY CONNECTICUT, Indiana, Kansas, and New York regulate hazardous substances in household products, according to a study conducted by the Committee on Toxicology and the Law Department of the American Medical Association. Bernard E. Conley, Ph.D., secretary of the Committee on Toxicoloy, said the study was conducted in preparation for drafting a model chemical labeling law. A fall conference of interested parties in government, industry, and medicine has been planned to draft the model law which will then be submitted to legislative bodies.

"The proposed legislation is intended to reduce careless and ignorant handling of potentially harmful products in and around the home, in small businesses, and in other areas where control of overexposure to chemicals is not as efficient as in the manufacturing process," Conley said.

The law will require informative labeling, including listing of possibly harmful ingredients, their potentialities for harm, directions for safe use, and first aid instructions.

The study shows that at present all the states require labeling of narcotics; 93 percent, of drugs; and 85 percent, of pesticides. However, only 52 percent require labeling of caustics and 10 percent of industrial chemicals.

At the national level, there are several chemical laws which include the Federal Caustic Poisons Act of 1927; the Food, Drug, and Cosmetic Act of 1938; and the Insecticide, Fungicide, and Rodenticide Act of 1947. In addition, the Interstate Commerce Commission and the Post Office Department have regulations regarding labeling, uses, and transportation of chemicals.

Only 10 of 25 state caustic acid laws are similar to the Federal Caustic Poisons Act. The federal act itself is limited to only 12 caustic and corrosive acids and alkalies in specified concentrations, of which some are known to be hazardous in lower concentrations. In addition, many dangerous acids and alkalies are not even included in the law.

"Of the 46 states with drug laws, only 19 conform to the Federal Food, Drug, and Cosmetic Act of 1938, even though 40 percent of all drugs sold are confined to intrastate commerce," Dr. Conley said.

"By and large there is greater agreement between state and federal laws in the area of pesticides than in any other major class of chemical products. Forty-three states have laws governing the sale and distribution of pesticides," he reported.

All but four states have poison laws or regulate the sale of poisons in some way. Only five states (California, Illinois, New Jersey, New York, and Oregon) require precautionary labeling of chemical products used in industrial establishments. Other states have special laws regulating specific individual chemicals. In fact, there are 16 types of these special laws, and some states have as many as 5 such statutes.

This article has been reprinted, condensed as indicated, from Pediatrics, *vol. 18, No. 6, pp. 943-957, December 1956.* Pediatrics *is published for the American Academy of Pediatrics, Evanston, Ill., by Charles C. Thomas, Publisher, Springfield, Ill.*

The Exposure of Children to Lead

By J. Julian Chisolm, Jr., M.D. and Harold E. Harrison, M.D.

Department of Pediatrics, School of Medicine, Johns Hopkins University, Harriet Lane Home of Johns Hopkins Hospital, and Pediatric Division of Baltimore City Hospitals, Baltimore, Md.

THE AVAILABILITY of a new and effective therapeutic agent has resulted in renewed interest in lead intoxication in childhood. . . .

* * * * *

. . . The purpose of this report is to . . . discuss the relative role which each of the several environmental factors may play in the production of lead intoxication in children. It is hoped that this may facilitate the implementation of adequate preventive measures.

Clinical Material and Diagnostic Criteria

The clinical material was derived from the pediatric clinics of the Harriet Lane Home of Johns Hopkins Hospital and of Baltimore City Hospital. In all, 197 children exposed to lead, including 89 patients with acute lead encephalopathy, have been studied. All dwelled in the urban slum areas of Baltimore. Within this group, 59 children were personally studied by the authors. In these children, observations on developmental status and personal-social home situations were made. The study was conducted during the years 1952 to 1954, inclusive.

An important part of the present survey was the detailed analysis of exposure factors in nine households in which abnormal sources of lead were demonstrated. Within these nine environmental units, there were 9 index cases and 17 other children under 6 years of age who served as controls, or a total of 26 subjects with known exposure to lead. Among the 17 control subjects of this household-study group, the incidence of unsuspected "secondary" cases was determined.

Another group of 33 children were patients who were studied during and after hospitalization for acute lead intoxication or encephalopathy during 1952-1954. In addition, a review was made of all hospital records of children admitted to the above clinics during the previous 12 years and indexed as "lead poisoning" or "lead ingestion." Among these records, 138 contained sufficient information to establish and classify the clinical diagnosis according to the criteria listed below. These constitute 138 of the 197 subjects included in this report.

DIAGNOSTIC CLASSIFICATIONS: All subjects are classified in six clinical categories. . . .

* * * * *

The numbers of subjects in each diagnostic category were as follows:

I. Exposed-normal (with presumed lead ingestion)	41
(a) record of lead source available	14
(b) record of lead source not available	27
II. Asymptomatic, increased lead absorption	39
III. Lead intoxication without encephalopathy	16
IV. Lead encephalopathy, mild	48
V. Lead encephalopathy, severe	41
	185

The remaining 12 subjects of the total group of 197 children exposed to lead were normal control subjects in the household-study group who apparently had not ingested lead-containing materials.

* * * * *

In all suspected cases of lead intoxication in children in Baltimore, samples of whole blood and of suspected environmental sources of lead were routinely analyzed for lead by the Baltimore City Health Department. . . . Samples of paint chips, painted plaster, etc., were collected in the homes by a visiting public health nurse. The lead content was estimated by a semiquantitative technique[11] [see page 32] and reported qualitatively as follows:

Lead absent—less than 0.1 percent of lead

Trace of lead—0.1 percent to 1.0 percent of lead

Positive for lead—1 percent to 5 percent of lead

Strong positive for lead—greater than 5 percent of lead

Results

In 9 households selected for detailed study of exposure factors there were 9 index cases and 17 other ambulatory children less than 6 years of age, who served as controls. Of the 9 index cases, 4 had acute lead encephalopathy, 2 had lead intoxication and 3 were classified as asymptomatic, increased lead absorption. . . .

* * * * *

An unexpected finding of this household-study was the discovery of 5 additional cases of unrecognized increased lead absorption and lead intoxication among the 17 control subjects. All of these unsuspected cases fell within the same age range as the index cases: namely, 12 to 35 months of age. No evidence of increased lead absorption or ingestion was demonstrated among the older

housemates. . . . Indeed, if the index cases are included, 14 of the 15 children aged 12 to 35 months in the 9 households were found to be ingesting potentially toxic quantities of lead.

* * * * *

In the entire series, records of identified sources of lead were available for 105 children. The locations of these sources about the dwellings are shown in Table V. Of the 220 samples tested which contained greater than 1 percent of lead, only 4 were obtained from cribs and furniture. More striking is the fact that 116 of the 220 sources were windowsills and frames. Peeling wallpaper and crumbling plaster accounted for an additional 48 sources. For each of the 105 exposed children, from 1 to 5 different sources containing greater than 1 percent of lead were identified among the various items each child was thought to have chewed. For 102 of them there was at least one source containing greater than 5 percent of lead. The lead was contained in the paint on the surfaces. The paint was old and flaking and frequently contained many layers, one or more of which may have contained lead pigments in which the content of lead may have been 30 to 70 percent of the total solids in the paint.

An attempt was made to ascertain the duration of exposure likely to produce toxic symptoms. Maternal estimates of the duration of their child's ingestion of lead-containing materials frequently were either unobtainable or unreliable. The type of exposure, however, permitted an indirect estimate of the probable duration of exposure, if the following assumptions were made: a) the locations of the sources of lead required that the children be ambulatory in order to reach them, and b) exposure began at the time of ambulation, or at 12 months of age on the average. With these assumptions the probable duration of exposure could be estimated as the interval in months between the child's first birthday and the age at which the child was classified as asymptomatic, increased lead absorption, or clinical lead poisoning was discovered. When the data were analysed on this basis, no statistically significant correlation could be found between presumed duration of exposure and severity of disease (exposed-normal children excluded). Indeed, the data indicated that the presumed exposure was of similar duration in all diagnostic classifications manifesting increased absorption of lead, with or without symptoms.

An explanation for this became apparent when the seasonal factor was taken into account. Thirty-two patients in the series developed acute lead encephalopathy prior to 2 years of age. The relationship between the seasonal factor and the presumed duration of exposure is shown for each of the patients in Figure 1, in which the month of the child's first birthday is joined by a bar with the month of onset of acute encephalopathy. The length of each bar, therefore, represents the assumed duration of exposure in months for each child. In the upper portion of Figure 1 the children passing their first birthday between May and September are shown. They were presumed to have been ingesting lead during some part of this 5-month summer period (when the peak incidence of acute lead encephalopathy occurs), yet they did not become ill until the summer of the following year as they approached 24 months of age. In the lower portion of Figure 1 are shown those patients reaching their first birthday during the winter months prior to April. They became ill during the first summer after the presumed onsets of ingestion.

Reading from top to bottom of Figure 1 the probable duration of ingestion, therefore, becomes progressively shorter and approaches a minimum of 3 months. Within this group of 32 patients there was no statistically significant correlation between month of first birthday and severity of, or survival during, acute lead encephalopathy. Excluding December, the distribution of births by month in the group does not differ significantly from the monthly distribution of births in Baltimore for the years 1951 to 1953 inclusive, during which most of the patients were born. In this small group the peak in births during December, and the passage of the older infants through their first summer of ambulation without symptoms, suggests that 5 to 6 months of lead ingestion may elapse prior to the onset of acute lead encephalopathy.

TABLE V

ENVIRONMENTAL SOURCES OF LEAD*

Location	Lead Content	
	Less than 1%	*1% or More*
	Interior Sources	
Windowsills & frames	3	116
Interior walls		
painted paper	38	16
painted plaster	24	32
Door frames	1	15
Furniture	3	3
Cribs	3	1
	Exterior Sources	
Door frames	3	10
Porches & housewalls	6	20
Fences & other walls	2	7
	Totals	
Interior sources	74	183
Exterior sources	11	37
	85	220

* The distribution, by lead content and location, of the 305 identified possible environmental sources of lead to which 105 subjects in this series were exposed. These sources were identified by analysis of paint scrapings taken from the various surfaces in and about the homes which gave evidence of having been chewed. For each subject one to seven such sources were identified. For all 105 subjects at least one source containing greater than 1% of lead was found. For 102 of the 105 subjects at least one source containing greater than 5% of lead was found.

The possibility that a minimum exposure period of 3 months is required, after which the advent of summer becomes the controlling factor in the production of symptoms, is also suggested by the following finding. In the entire group there were 14 children with unequivocal evidence of excessive lead ingestion including identification of at least one environmental source containing greater than 1 percent of lead, but with no evidence of increased lead absorption. Eight of these 14 exposed-normal children were less than 15 months of age. Conversely, among the 89 patients with encephalopathy, the youngest was 15 months of age.

Although these data on duration of exposure were indirectly deduced, they are supported by histories ob-

tained from many parents, who stated that they had seen their children ingesting paint flakes or had found plaster particles in the child's stools since the time the child had begun to walk. It was also observed that among patients who survived an initial episode of acute lead encephalopathy and were reexposed to lead, recurrent acute episodes of encephalopathy usually did not occur until the following summer.

An analysis of the occurrence of severe permanent damage to the brain among survivors of an initial attack of acute lead encephalopathy indicates the importance of continued environmental exposure to lead in increasing the incidence of central nervous system sequelae. In the entire series 61 survivors of acute lead encephalopathy were treated more than 12 months prior to evaluation of sequelae. . . . Fifteen of the group were lost from follow-up. Forty-six of the 61 patients . . . have been followed for 1 year or longer. Of these 46 patients, 14 were known to have continued ingestion of lead for periods of 2 months or longer, 4 had doubtful reexposure, and 28 had no known reexposure to lead after the initial episode of encephalopathy.

The incidence of sequelae in the survivors of acute lead encephalopathy was analysed, without regard to type of therapy during the acute phase, to determine whether there was any association between the occurrence of severe permanent residua in the central nervous system and reexposure to lead. Presence of one or more of the following manifestations was considered evidence of serious permanent damage to the brain: severe mental retardation, recurrent convulsions, and severe behavior disorder. Of the 23 survivors classified as having these "severe sequelae," 6 demonstrated one of these criteria, 5 had two and 12 fulfilled all three. . . . There is, statistically, a highly significant association between the occurrence of such "severe sequelae" and reexposure to lead following recovery from mild encephalopathy. . . . The correlation between sequelae and reexposure to lead is more difficult to assess in the group surviving an episode of severe encephalopathy. Among the 20 severe cases, reexposure was uncertain in 4. . . . If these doubtfully reexposed cases are considered as not re-exposed . . . reexposure is not statistically significant. For the entire group of survivors of mild and severe encephalopathy there is no significant correlation between sequelae and the duration of acute encephalopathy, type of therapy during the acute phase, or concentration of lead in blood after the acute illness. The loss of patients from follow-up does not vitiate the analysis. In survivors not reexposed to lead there is no correlation between sequelae and age of incidence at the 18, 24, or 30-month levels.

RELATIONSHIP BETWEEN MONTH OF FIRST BIRTHDAY AND MONTH OF INCIDENCE OF ACUTE ENCEPHALOPATHY

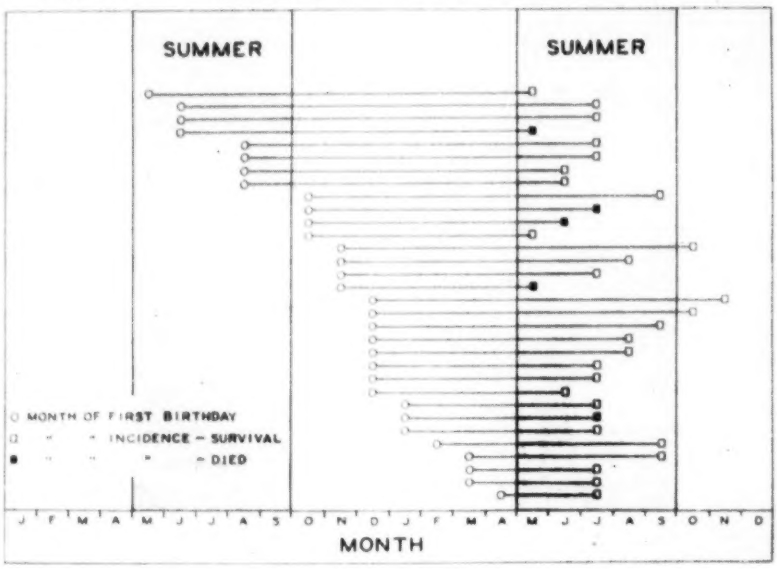


Fig. 1. In this graph the month of onset of acute symptoms for each of the 32 patients* who developed acute lead encephalopathy prior to 2 years of age is shown on the right. On the left is the month of the child's first birthday. These points are joined by a bar. The length of each bar, therefore, represents the assumed duration of lead exposure (in months) for each child. Twelve months of age was selected as the average age of ambulation. The nature of the exposure in this series required that the child be ambulatory in order to reach the sources of lead in his environment. In this manner a relation between presumed duration of exposure to lead and the seasonal factor in the production of symptoms can be demonstrated.

*The second and third bars of the chart should terminate in June rather than July of the year following the first birthday.

Discussion

. . . Eighty-eight of the 105 cases of acute lead intoxication occurred during May, June, July, August, and September. Ninety of the 105 cases occurred in children between the ages of 12 and 36 months. It need only be re-emphasized here that homes in which the children of this series lived had several features in common: they were old and in varying states of deterioration and disrepair. In the homes visited, crumbling, painted plaster or loose paint chips from walls, ceilings, door frames, window-sills, outside porches, and fences were readily accessible to the hands and mouths of inquisitive small children. The data of the present study show the intensity of this type of exposure; but they cannot be applied directly to such questions as the minimal quantity of lead toxic for children or the maximal safe content of lead in liquid paint.

Among the environmental data presented, two factors stand out:

tained from many parents, who stated that they had seen their children ingesting paint flakes or had found plaster particles in the child's stools since the time the child had begun to walk. It was also observed that among patients who survived an initial episode of acute lead encephalopathy and were reexposed to lead, recurrent acute episodes of encephalopathy usually did not occur until the following summer.

An analysis of the occurrence of severe permanent damage to the brain among survivors of an initial attack of acute lead encephalopathy indicates the importance of continued environmental exposure to lead in increasing the incidence of central nervous system sequelae. In the entire series 61 survivors of acute lead encephalopathy were treated more than 12 months prior to evaluation of sequelae. . . . Fifteen of the group were lost from follow-up. Forty-six of the 61 patients . . . have been followed for 1 year or longer. Of these 46 patients, 14 were known to have continued ingestion of lead for periods of 2 months or longer, 4 had doubtful reexposure, and 28 had no known reexposure to lead after the initial episode of encephalopathy.

The incidence of sequelae in the survivors of acute lead encephalopathy was analysed, without regard to type of therapy during the acute phase, to determine whether there was any association between the occurrence of severe permanent residua in the central nervous system and reexposure to lead. Presence of one or more of the following manifestations was considered evidence of serious permanent damage to the brain: severe mental retardation, recurrent convulsions, and severe behavior disorder. Of the 23 survivors classified as having these "severe sequelae," 6 demonstrated one of these criteria, 5 had two and 12 fulfilled all three. . . . There is, statistically, a highly significant association between the occurrence of such "severe sequelae" and reexposure to lead following recovery from mild encephalopathy. . . . The correlation between sequelae and reexposure to lead is more difficult

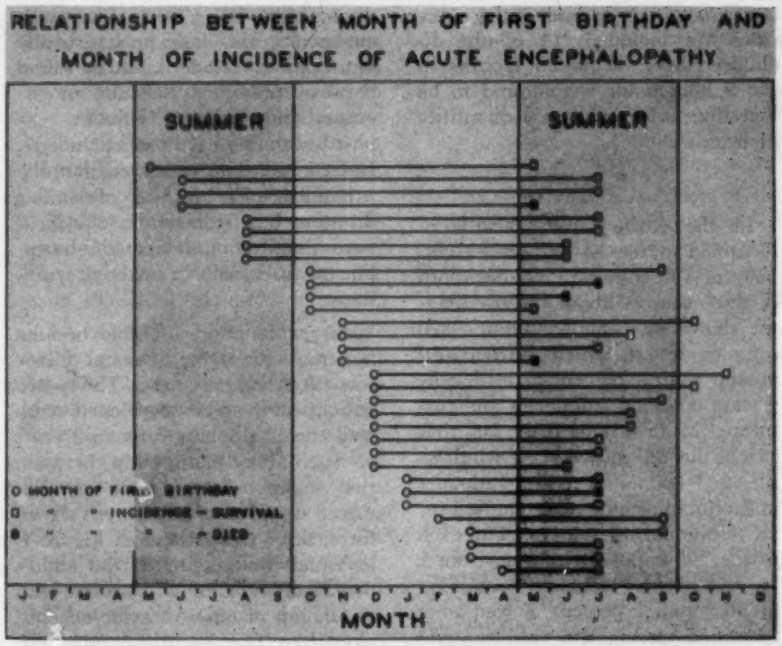


Fig. 1. In this graph the month of onset of acute symptoms for each of the 32 patients* who developed acute lead encephalopathy prior to 2 years of age is shown on the right. On the left is the month of the child's first birthday. These points are joined by a bar. The length of each bar, therefore, represents the assumed duration of lead exposure (in months) for each child. Twelve months of age was selected as the average age of ambulation. The nature of the exposure in this series required that the child be ambulatory in order to reach the sources of lead in his environment. In this manner a relation between presumed duration of exposure to lead and the seasonal factor in the production of symptoms can be demonstrated.

*The second and third bars of the chart should terminate in June rather than July of the year following the first birthday.

to assess in the group surviving an episode of severe encephalopathy. Among the 20 severe cases, reexposure was uncertain in 4. . . . If these doubtfully reexposed cases are considered as not re-exposed . . . reexposure is not statistically significant. For the entire group of survivors of mild and severe encephalopathy there is no significant correlation between sequelae and the duration of acute encephalopathy, type of therapy during the acute phase, or concentration of lead in blood after the acute illness. The loss of patients from follow-up does not vitiate the analysis. In survivors not reexposed to lead there is no correlation between sequelae and age of incidence at the 18, 24, or 30-month levels.

Discussion

. . . Eighty-eight of the 105 cases of acute lead intoxication occurred during May, June, July, August, and September. Ninety of the 105 cases occurred in children between the ages of 12 and 36 months. It need only be re-emphasized here that homes in which the children of this series lived had several features in common: they were old and in varying states of deterioration and disrepair. In the homes visited, crumbling, painted plaster or loose paint chips from walls, ceilings, door frames, window-sills, outside porches, and fences were readily accessible to the hands and mouths of inquisitive small children. The data of the present study show the intensity of this type of exposure; but they cannot be applied directly to such questions as the minimal quantity of lead toxic for children or the maximal safe content of lead in liquid paint.

Among the environmental data presented, two factors stand out:

the intensity of lead exposure provided by a small quantity of paint flakes and the role of the season in precipitation of acute lead encephalopathy.

* * * * *

. . . Under the sponsorship of the American Academy of Pediatrics, the Committee on Hazards to Children of the American Standards Association studied the problem and recommends that paints may be considered safe for use on children's toys and furniture and housing interiors if the lead content does not exceed 1 percent of the total solids.[19] Paints and other surface coatings meeting this specification and those relating to other potentially toxic metals may be labelled: "conforms to American Standard Z66.1-1955." It is essential to recognize that this recommendation applies only to the two or three layers of paint usually applied to a surface; not to multiple layers of paint as may be found in old, deteriorated housing. As the number of layers increases or when a layer of paint containing lead pigments is added, the quantity of lead per unit area increases to the point where even a small flake may contain an excessive amount of lead. . . .

In Table V the location and lead content of environmental sources of lead are shown; 102 of these 105 children were exposed to at least one source containing greater than 5 per cent of lead. No case of lead intoxication has been found where the only source of lead contained less than 1 percent of lead in the dried paint surface. The present data apply to old housing. Inasmuch as lead is presently used as a paint drier as well as a pigment, 1 per cent of lead in total paint solids approaches the minimum consistent with a satisfactory house paint. It is conceivable that poor maintenance of modern housing with resultant accumulation of many layers of paints (containing as little as 1 percent of lead) on surfaces accessible to children may in the future constitute a hazard to small children.

Evidence has been presented which indicates that, after a minimum of approximately 3 months of this type of exposure, the advent of summer (in the presence of continued exposure to lead) is the controlling environmental factor in the precipitation of acute lead encephalopathy (Fig. 1). . . .

When the probable duration of exposure exceeded 3 months, no correlation could be demonstrated between increasing severity of disease and further monthly increments in exposure. This finding may be explained by the interplay of two other factors: the magnitude of lead ingestion observed in the household-study group and the seasonal factor in the precipitation of acute symptoms. It emphasizes the urgency during the summer months of the prompt removal of children manifesting asymptomatic increased lead absorption from such intense exposure to lead. The data indicate that, in such a patient, acute encephalopathy may develop if exposure is permitted to continue but a few weeks longer.

* * * * *

The finding in the household-study of 5 "secondary" cases among the 6 control subjects aged 12 to 35 months is significant. If confirmed, it indicates that a larger public health aspect of this disease exists in old housing areas than has heretofore been recognized. Furthermore, wherever an index case is found, his environmental contacts under 3 years of age should be submitted to careful clinical and laboratory examination.

. . . a highly significant correlation between reexposure to lead and the incidence of severe permanent sequelae in the central nervous system in survivors of an initial episode of acute encephalopathy has not previously been reported. This indicates that immediate and absolute prevention of reexposure may, perhaps, be the single most important long-term factor in the eventual outcome in such patients. In our experience this has been satisfactorily accomplished only by burning the old lead-containing paint completely away or by transfer of the child to a new dwelling in good repair.

The term "pica" is frequently used in association with both lead poisoning and with mental defect in children. Thus, in the minds of many, plumbism and mental deficiency are frequently associated. This is not so with respect to the ingestion of other toxic substances, a single ingestion of which suffices to produce symptoms. The want of toddlers in the 1-year age group to taste and eat a large variety of foreign material is well known and is considered to be a part of their normal behavioral pattern, at least for a short span of time.

In the present study all members of the household group were submitted to psychometric examination. Within this group no striking developmental deviations could be found between the affected and the non-affected members. In addition, 28 patients who were not reexposed were psychometrically evaluated 1 year after recovery from acute lead intoxication and encephalopathy. The distribution of intelligence quotients on the basis of the revised Stanford-Binet Intelligence Scale, Form L, was as follows:

Intelligence Quotient	*Numbers of Patients*
60- 69	3
70- 79	2
80- 89	5
90- 99	9
100-109	5
110-119	4

These findings would agree with the concept that the ingestion of foreign material exhibited by these children was not a manifestation of mental deficiency, but that the ready availability of a highly toxic material in the environment was the important factor. This is certainly supported by the high "secondary" attack rate found among the control subjects of the household-study.

If the assumptions are made that flaking, lead-containing paints are widely distributed in old housing, that there are large numbers of infants and toddlers living in such a physical environment, and that normal as well as defective children may readily ingest amounts of lead sufficient to produce intoxication,

(To page 31)

THIS ARTICLE has been condensed for the *Home Safety Review* from a paper by Dr. Arena which was read in the Symposium on Childhood Poisons before the Joint Meeting of the Section on Pediatrics and the Section on Preventive and Industrial Medicine and Public Health at the 105th Annual Meeting of the American Medical Association, Chicago, Ill., June 12, 1956, and originally published in the *Journal of the American Medical Association*, vol. 163, No. 15, pp. 1341-1343, April 13, 1957.

PRACTICAL MANAGEMENT OF COMMON POISONING

By Jay M. Arena, M.D.
Department of Pediatrics
School of Medicine
Duke University,
Durham, N. C.

FOR MOST PHYSICIANS, not many days go by without an emergency telephone call from a frantic mother who says that her child has ingested some poisonous substance. This is understandable, however, when one realizes what agents the usual household contains today as compared to those agents hazardous to children 20 to 30 years ago. We now have deodorants, depilatories, detergents, herbicides, insecticides, rodenticides, petroleum products, stimulants, sedatives, analgesics, and so forth.

General Treatment

Orally Taken Poison.—When poisons have been taken by mouth, obviously it is important to remove the unabsorbed poison. The simplest procedure is to induce vomiting. As far as children are concerned, vomiting is best induced by having them drink a glass of milk after which they should be gagged with the finger. The use of warm saline solution or mustard water as an emetic is impractical in most instances since children refuse to drink this type of concoction. If the above procedure does not cause vomiting, gastric lavage should be instituted at once. The importance of emptying the stomach quickly cannot be overemphasized, for it is the essence of treatment of any type of poisoning and is often a lifesaving procedure. Unless the patient is comatose or in cases of poisoning due to petroleum distillates and corrosive agents, gastric lavage should be carried out in every instance.

Chemical Antidotes: Chemical substances that neutralize, precipitate, oxidize, or otherwise alter the poison to decrease its toxicity or lessen its absorption are best administered in the lavage fluid. Activated charcoal is capable of absorbing large amounts of alkaloids like strychnine, morphine, and atropine, as well as inorganic substances such as mercuric and arsenic compounds. Tannic acid precipitates many alkaloidal and metallic poisons. An infusion of a strong solution of tea may be employed, if powdered tannic acid is not available. Magnesium oxide is useful for neutralizing mineral acids. The "universal antidote" is a combination of activated charcoal, tannic acid, and magnesium oxide.

Inhaled Poisons.—In cases of gas poisoning, the first act should be to remove the victim from the presence of the gas and apply artificial respiration.

Injected Poisons.—If an injection of a poison has occurred, application of tourniquets central to the point of injection may slow the rate of absorption. Quantities of unabsorbed poison may have to be removed by means of surgery and by suction similar to that commonly advised for snakebite.

Supportive Measures Should Include: (1) Fluid Therapy. (2) Artificial Respiration. (3) Cardiac Therapy. (4) Shock Therapy. (5) Treatment of Convulsions.

Poisoning Due to Specific Agents

Barbiturate Poisoning. — The increased use of sedatives in this jittery age has converted practically every family medicine chest into a miniature barbiturate warehouse. In England and Australia, the new barbiturate antagonists have been found to be very effective in the treatment of acute and chronic cases of barbiturate poisoning. These drugs have also been found to be useful in the treatment and/or prevention of intoxication due to morphine.

Lead.—Lead may enter the body via the lungs, mouth, or skin. The most serious effects follow inhalation of lead. Cases of lead poisoning due to inhalation of fumes caused by

the combustion of storage battery casings have been reported frequently. Other causes are paint, lead toys, motor fuel, and lead water pipes.

Aspirin.—The death rate from poisoning with aspirin has increased from 30 to 130 persons a year in the past 20 years. Since the advent of "candy" aspirins in 1948, the incidence of aspirin poisoning has increased by leaps and bounds.

Boric Acid.—Boric acid poisoning has received considerable publicity of late, and there is no question that the indiscriminate use of boric acid over large areas of broken skin or mucous membrane is dangerous. However, the small amount present in baby powders, used mainly as a buffering and fungistatic agent, is safe for normal use. The symptoms of boric acid poisoning are erythema and exfoliation of the skin, vomiting, inanition, dehydration, and convulsions. Treatment consists of the removal of the source of boric acid, and the administration parenterally of fluids and barbiturates to control the convulsions.

Insecticides.—Insecticides of many types are found about the home. The agents, such as chlorophenothane (DDT), containing chlorinated hydrocarbon groups have been widely publicized, and the dangers are well known. However others, such as parathion, tetraethylpyrophosphate (TEPP), and hexaethyltetraphosphate (HETP), containing the organic phosphate group are highly toxic, and their dangers are not well recognized.

Gastrointestinal disturbances, sweating, salivation, miosis, involuntary defecation and urination, confusion, muscle weakness, and generalized convulsions comprise the list of symptoms of poisoning by insecticides.

Atropine, although specific in its action against the parasympathetic effects of certain insecticides, does not help the muscular weakness. Therefore, the use of artificial respiration may be required for many hours. Suction to clear the airways and oxygen given under positive pressure should be used if pulmonary edema occurs. In such cases, the use of a respirator may prove to be lifesaving. Respiratory depression contraindicates the use of morphine. Pronounced changes in electrolyte levels and serum pH may require the administration of adequate amounts of calcium and sodium bicarbonate.

Kerosene.—A common type of poisoning in children in the South is that due to the ingestion of kerosene, which is sometimes left carelessly in the house in a soft drink bottle. Thirsty toddlers sometimes sample the liquid. In the treatment of poisoning due to the ingestion of kerosene, the millenium perhaps has not been reached, but the bulk of the experimental and clinical evidence would indicate that the use of emetics and gastric lavage are contraindicated. The risk of aspiration and pneumonitis far outweighs the occasional toxic manifestation and involvement of the central nervous system due to gastric absorption. The administration of antibiotics (to prevent the development of bacterial pneumonia) should be started early.

Moth Repellents.—Mothballs and moth repellent flakes and cakes most frequently contain *p*-dichlorobenzene. Since halogenation of aromatic compounds decreases toxicity, ingestion of *p*-dichlorobenzene rarely causes poisoning, but prolonged exposure to its fumes will cause damage to the liver and cataracts. Some moth repellents contain naphthalene alone or combined with *p*-dichlorobenzene. A few contain camphor. As little as 2 gm. of naphthalene has caused fatal poisoning in a child. However, the fatal dosage of this repellent in most cases is considerably larger.

Naphthalene poisoning should be treated with gastric lavage or the administration of emetics. Fluids should be given in large amounts orally and parenterally to decrease the concentration of the poison in the urine. During convalescence from severe intoxication, the diet should be high in carbohydrates and vitamins, moderately high in protein, but low in fats to promote the regeneration of liver tissue. Several cases of hemolytic anemia have been reported after the ingestion or exposure to this agent.

Fluoride.—Intoxication due to sodium fluoride or sodium fluosilicate (Na_2SiF_6) results from the ingestion of certain commercially available roach or ant powders. Ingestion of appreciable amounts of sodium fluoride causes death within eight hours unless the patient is promptly treated.

Carbon Monoxide.—Carbon monoxide is a gas with approximately 300 times the affinity of oxygen for hemoglobin. It decreases the rate of dissociation of oxygen and hemoglobin. Less oxygen is present in the blood, and a smaller proportion of oxygen is freed in the tissues. It, therefore, produces anemia and anoxia. The administration of oxygen with the use of a mask hastens the removal of the poison.

Prevention

Since incidents of poisoning usually involve children under 5 years of age, the incidents are due to someone's carelessness and neglect. It is paramount, then, in prevention to protect these children. Parents, therefore, need to be educated to an understanding of the changes that have made the American home a hazard instead of a haven. Physicians need education, too, so that they may be alert to the new toxic drugs or household agents. Manufacturers of drugs and household agents also should be made aware of their responsibilities (i.e., to furnish more informative labeling and so forth) in the prevention of these tragic accidents.

Summary

Not many days go by without most physicians' getting an emergency call from a frantic mother stating that her child has ingested some poisonous substance. The alarming feature of this problem is the regularity with which various household agents and drugs are swallowed by children. This is understandable, however, when one realizes that there are over a quarter of a million different trade name substances on the market today. The general treatment as well as specific therapy for poisoning by some of the common types of agents is, therefore, important.

1410 Duke University Rd.

Inservice Training for the PHN

By Mrs. Mary K. Foster, M.P.H.
Public Health Educator
Rochester Health Bureau
Rochester, N. Y.

In-Service Training Sessions Re Safety Education for Public Health Nurses

How These Training Sessions Started

Various members of the Health Bureau are active in safety education activities in the community. Those featured in these sessions are the public health educator who has been a member of the Home and/or Child Safety Committees of the Rochester Safety Council since 1949, served as chairman of the Home Safety Committee and has been a member of the Board of Directors of the Council and two of the 5 supervising public health nurses who also serve on these committees.

The Supervising Public Health Nurse who [was] on the Home Safety Committee during 1955-56, with the Public Health Educator had several discussions regarding the role of the staff Public Health Nurse in home safety: How she could do more in home safety; what can be done to assist her do a more effective job in home safety teaching. A plan evolved to suggest to the Director of Nurses and her supervisory staff that sessions on (Home) safety be included as part of the Public Health Nurse's In-Service Training Program. This supervising public health nurse cleared this suggestion with the Director of Nurses and approval was given to conduct the sessions at the end of June and early July.

Planning

The supervising public health nurse on the Child Safety Committee was also assigned to the planning committee for these sessions. The public health educator served as consultant to the two supervising public health nurses in planning and conducting these sessions. She also coordinated the entire sessions. Several conferences were held before the final plans were made. (Attached outline gives details [see below]).

Since time was a limiting factor, we agreed

—To conduct two sessions (total 5 hours—2½ hrs. each afternoon) and devote most of the time to home safety and cover as much of school and community safety as time permitted.

—That the two supervisors would

—Decide the make-up of each group. It was agreed to have about 25 nurses in each group—some new staff, some with few years experience and some with a longer service record.

—Send out questionnaires to Public Health Nurses to find out their interests and needs in safety education.

—Each one of these supervisors prepared herself for her part in the sessions (Note outline)

—That the public health educator would

—Prepare the chalk talk on safety and demonstrate how to use this teaching method and explain how to prepare a chalk talk . . . (Complete talk available in this Office of Health Education)

—Demonstrate how to use slides, photographs, film and printed materials as effective teaching aids.

—Prepare . . . mimeographed materials.

—Locate and utilize community safety resources. (Ex. Fireman and House of Hazards)

—Coordinate the sessions.

Since this was the first time such a training session was conducted the Public Health Educator played a dominant, active role—on a demonstration basis—especially because teaching methods and teaching materials were demonstrated.

(The above is a general description of how this was done. Anyone interested in more details can confer with this Public Health Educator.)

* * * * *

For this inservice training, the following bibliography was prepared for public health nurses by Mrs. Foster.

TO: Public Health Nurse
FROM: Public Health Educator, Mary Foster
ITEM: Reference Material on Safety

The following suggestion and reference materials on home safety relate to:

1. General information and current statistics.
2. The role of the nurse in home safety.

These materials are in the Rochester Health Bureau, Office of Health Education. Most of these references are in the *bound periodicals.* These bound periodicals are *REFERENCE BOOKS* and are to be used only in the Office of Health Education—*they are not to be taken out of the Office of Health Education.*

In addition to these references, there are:-

1. Pamphlets in the pamphlet room that are for public distribution and
2. Books that you may borrow for a week or two.
3. Reference file of materials available at the local Safety Council.
4. New York State Health Department—Film and Exhibit Catalogue which includes Safety Exhibits and Films.
5. Reynolds Film Library Cata-

THE ROCHESTER Health Bureau, Rochester, N. Y., in 1956 developed an inservice training program for public health nurses to learn about home safety. Mrs. Mary K. Foster, M.P.H, public health educator with the bureau, has reported on this as follows in an article "In-Service Training Sessions Re Safety Education for Public Health Nurses" in the September 17, 1956 issue of a bulletin *Have You Heard . . .* which was included with her 1956 Home Safety Inventory report. The bulletin was published by Mrs. Mary Armstrong, Education Consultation Section, Office of Public Health Education, New York State Department of Health, Albany, N. Y.

logue (Main Library) which includes many films on safety.

General Information on Home Safety

1. *Home Accident Prevention, A Guide for Health Workers,* U. S. Department of Health, Education and Welfare—a working guide for professional public health workers. It is an outline only and must be supplemented by local facts and references on methods. (Although published in 1953 the same problems are still with us!)
2. *Home Safety Review*—National Safety Council—published monthly [except July and August] for "home safety program leaders." The value of this reference depends upon the responsibility of the nurse for home safety beyond incorporation of education and accident reporting or follow-up in her routine duties. Many current programs and suggested activities are reported in this magazine.
3. *Home Safety Inventory 1955*[1] —published by the Home Safety Conference of the National Safety Council.

   Programs of local health departments throughout the United States are briefly reported here and might include new approaches of interest to nurses. (Reports of many other groups also included.)
4. *Accident Facts, 1955*[2] *Edition* —published by National Safety Council. National statistics: trends, types of accidents by age, costs, etc.
5. *Public Health Reports*
   a. Sept. 1954; Vol. 69, No. 9, pp. 844-48, Report of Southern Branch, APHA Meeting, 1954.
   b. Feb. 1955; Vol. 70, No. 2, pp. 220-3, Reports of APHA Meetings on "Childhood Accident Prevention," 1954.
   c. June 1955; Vol. 70, No. 6, —"An Experience in Home Injury Prevention," Wain, Samuelson and Hemphill.
   d. Oct. 1955; Vol. 70, No. 10, "The Illusive Phenomena in Accident Proneness"—Wilse B. Webb.
6. *American Journal of Public Health*
   a. Jan. 1954; Vol. 44, No. 1 —"Home Safety and Accident Prevention in a Child Health Conference"—Jacobziner and Rich.
   b. June 1954; Vol. 44, No. 6 —Editorial, "Accidents in the Factory and in the Home."
   c. Oct. 1954; Vol. 44, No. 10 —"Public Health's Interest in Home Accident Prevention," Emil A. Tiboni.
   d. Dec. 1954; Vol. 44, No. 12 —"A Poisoning Control Program," Edward Press, M.D., M.P.H., and Robert B. Mellins, M.D.
   e. April 1955; Vol. 45, No. 4 —"An Educational Project in Childhood Accident Prevention," Renee Zindwer, M.D., M.P.H. and "Discussion: Safety Education Without Fear," Paul Lemkau, M.D.
   f. Oct. 1955; Vol. 45, No. 10 —"Epidemiologic Principles Applicable to the Study and Prevention of Child Accidents," McFarland, R.A.
   g. Jan. 1956; Vol. 46, No. 1 —Editorial—"Should the Health Officer Look at the Automobile—The Misguided Missile?"—Appropriate incentive for home safety also.
7. *Health News,* New York State Department of Health—May 1954; Vol. 31, No. 5—Accident Prevention issue.
8. "Getting Down to Brass Tacks" —Metropolitan Life Insurance Company—Booklet describing a home safety demonstration.
9. *New York State and Local Statistics*

   The "Home Safety Kit" provided to all local health departments in October, 1954, contains several pieces of material (including #1 listed above). Among these was "Bringing Home Safety Home." This booklet summarized state statistics by type, causes of death by age and fatal accidents by type and place of occurrence, 1950-52. The current data (1950-54) is now being compiled for a revision of this booklet. (1950-54 statistics for Rochester [were] attached).

Role of the Nurse

1. *Public Health Reports*—Vol. 69, No. 9, Sept. 1954, pp. 846-

[1]The 1956 report is now available, Ed. note.

[2]The 1957 edition is now available, Ed. note.

7—"Safety Promotion Is Part of Nurse's Daily Work," Madelyn H. Davis, B. S.—Georgia Department of Public Health. (Also listed in "General Information").

2. *Nursing Outlook*—National League for Nursing
   a. March 1953; Vol. 1, No. 3—"A Home Accident Survey," Marion Wetzel.
   b. July 1953; Vol. 1, No. 7—Editorial, "Developing Immunity to Accidents."
   c. Nov. 1953; Vol. 1, No. 11—"The Concept of Accident Proneness"—An abstract from *Industrial Medicine;* 22: 141-148, April, 1953.
   d. Dec. 1953; Vol. 1, No. 12—"Preventing Home Accidents," Florence Genschmer.
   e. Dec. 1954; Vol. 2, No. 12—"A Course for Baby Sitters," Coburn, Miczynski and Van Denend—Includes in the course plan, emphasis on safety.
   f. Nov. 1955; Vol. 3, No. 11—"The Nurse's Role in Accident Prevention," Marian Rolen—Refers to hospital nurse—but applicable in part to public health.)
   g. Feb. 1956; Vol. 4, No. 2—"The Home Accident Prevention Nursing Consultant," Pershing and Davis.
3. *The American Journal of Nursing,* American Nurses Association
   a. June 1955—Vol. 55, No. 6—"Home Accidents Resulting from Gas," Keehn, Robert J.
   b. July 1955—Vol. 55, No. 7—"The Industrial Nurse and Home Accidents," Barbara J. Smith—Reference to motivation, home visits and cooperation with others.
   c. Oct. 1955—Vol. 55, No. 10—"Nurses Help Prevent Accidents in Homes," Shepard, William P.—Special reference to older people.
4. "The Role of Public Health Workers in Home Accident Prevention"—Home Safety Unit, Georgia Department of Public Health.

Below is an outline used for the inservice training of public health nurses by the Rochester Health Bureau, Rochester, N. Y.

OUTLINE OF IN-SERVICE TRAINING SESSIONS REGARDING SAFETY EDUCATION FOR PHN'S

TIME AND PLACE

Two sessions, (total 5 hrs. - 2½ hrs. each afternoon), will be held June 26th and 27th and July 2nd and 3rd at the Health Bureau starting at 2:30 P.M. about 25 people in each group.

WHAT WE AIM TO DO

1. Review the safety problem, primarily in the home (and in the school - to the extent that time permits).
2. Point out that safety teaching is a part of the PHN's regular job.
3. Review how the PHN does safety teaching in the home, school and community.
4. Explain the "live and learn theory" of accident prevention: relationship between protection and education.
5. Demonstrate some teaching methods PHN's use in safety education work:-
   - -Chalk talk and lecture
   - -Buzz session
   - -Role play
   - -Discussion
6. Demonstrate how to use available visual aids in field of safety.
   - -Home safety slides
   - -House of hazards
   - -Film - Mrs. Hazard's House
   - -Pamphlets
   - -Photographs
7. Point out some of the safety materials available in the Office of Health Education, Rochester Health Bureau and in the community (Rochester Safety Council, Rundel Film Library, etc.)

PLAN FOR THE 2 SESSIONS

1st session

SUBJECT CONTENT	METHOD	PERSON IN CHARGE
1. Home Area Accidents -what are they -causes -the figures	Chalk Talk -Pages 1-15 & -Pages 44, 45, 48 - 54 Discuss mimeographed sheets Causes of accidents	Mary Foster
2. How do you as a Public Health Nurse Do Safety Teaching -In the Home -In the School -In the Community	Buzz sessions - about 12 minutes 3 groups 7 or 8 in a group	Margaret Cook - Home Group Celeste Kenney - School " Mary Foster - Community " (each group will have a leader and recorder selected by Margaret Cook & Celeste Kenney)

SUBJECT CONTENT	METHOD	PERSON IN CHARGE
3. Hear and write on blackboard report from the "Home Group"	Discussion based on PHN's report from buzz session	M. Cook - Discussion Leader C. Kenney - Recorder on board.
4. Continue topic "How Do You As A PHN Do Safety Teaching in the Home" -Give supplemental material in addition to buzz session	Combination of:- Lecture Role play home visit where PHN does home safety teaching Demonstrate use of a pamphlet on safety in this visit. Safe At Home Discussion based on role play situation -Pass out mimeo sheet "Let's Begin With The Most Dangerous Place" prepared by Office of Health Education	M. Cook
5. Break		
6. Dietrich's live and learn theory	Discussion and chalk talk, P. 16 - 20 Introduce the film - P. 21 "Mrs. Hazard's House" Show the film Discuss the film -Introduce the idea of using a check list, ex. SAFE AT HOME, as a basis for discussion in a group meeting Continue with chalk talk -live & learn theory -Protection -Education -Learning -Summary (P. 22 - 43) (P. 46 - 47)	Mary Foster
2nd Session 1. Review of 1st session Introduce 2nd session	Report	Mary Foster
2. Hear and write on blackboard report from buzz groups on School and Community	Discussion based on PHN's report from the 2 groups - school and community	C. Kenney - Leader M. Cook - Blackboard Recorder
3. How do you as a PHN Do safety teaching in the school and community -Give supplemental material in addition to report from buzz session	Combination of:- Lecture Discussion Role Play (on the spot) Demonstration -Use of Home Safety Slides for a group meeting	C. Kenney
4. Break		
5. Safety resource in the community a. House of Hazards	Demonstration & Discussion	Jack Herman - Fire Prevention Bureau
b. Kit of Safety Materials available in the community	Discussion of Materials	Mary Foster
c. Exhibit of photographs	Discussion	Mary Foster
6. Comments regarding National Safety Inventory and part Public Health Nurse will play	Discussion	Mary Foster
----	General Discussion --	
	End of Session	

MRS. MARJORIE BEGLEY, antidote chart chairman, El Monte Community Safety Council, El Monte, Calif., states that originally 13,000 copies of this chart were distributed to local school children. Last year the chart was recommended in the nationally-syndicated column of Walter C. Alvarez, M.D. Last November the *California Parent-Teacher* magazine reprinted the chart in full as their center spread, and in the April 1957 issue of *Action for Safety,* a publication of the National Education Association, the chart was publicized.

Poison Antidote Chart

Antidotes for Common Household Poisons

Developed by EL MONTE COMMUNITY SAFETY COUNCIL, EL MONTE, CALIFORNIA

Member of the National Safety Council

UNIVERSAL ANTIDOTE

IF YOU DO NOT KNOW WHAT VICTIM HAS SWALLOWED, as substance is not listed on this chart, USE THE UNIVERSAL ANTIDOTE, a general antidote for a large number of poisons. Every household should have this mixture on hand at all times. Ingredients may be purchased at your drug store.

Mix together: 2 tablespoons pulverized charcoal
1 tablespoon magnesium oxide
1 tablespoon tannic acid

Dosage: Mix 1 heaping teaspoonful in a small glass of water and MAKE the victim drink it.

ATTENTION

When a person swallows poison, you are faced with an emergency. The first thing you must do is to CALL A DOCTOR. If you cannot locate a doctor immediately, follow the instructions on this chart.

REMEMBER THAT YOU MUST ALWAYS DILUTE THE POISON BY GIVING LARGE AMOUNTS OF LIQUID.

THIS CHART WAS CHECKED BY THE BUREAU OF HEALTH EDUCATION OF THE AMERICAN MEDICAL ASSOCIATION

ACETONE — large quantity warm water, induce vomiting.

ALCOHOL — baking soda solution, induce vomiting.

AMMONIA — do NOT induce vomiting. Give grapefruit juice, or 1 part vinegar to 3 parts water. Follow with milk.

ANT POISON (containing arsenic) — large quantity warm salt water, induce vomiting. Follow with large dose castor oil and milk.

ARSENIC — large quantity warm salt water, induce vomiting. Follow with large dose castor oil and milk.

ASPIRIN — Universal Antidote, induce vomiting.

ATROPINE — strong tea or tannin, induce vomiting.

BARBITURATES — large quantity warm salt water or baking soda solution, induce vomiting. Follow with strong coffee.

BENZENE — large quantity warm water, induce vomiting; Universal Antidote.

BICHLORIDE OF MERCURY — large quantity warm water, induce vomiting. Follow with egg whites in milk.

BLACKLEAF 40 — strong tea or tannin, induce vomiting.

BLEACH, HOUSEHOLD — cooked cereal followed by olive oil or salad oil.

BORDEAUX MIXTURE (copper sulphate) — large quantity warm water, induce vomiting. Follow with egg whites in milk.

CAMPHOR — large quantity warm water, induce vomiting; Universal Antidote.

CARBOLIC ACID — large quantity warm water containing epsom salts, induce vomiting.

CARBON TETRACHLORIDE — large quantity warm water, induce vomiting. Universal Antidote.

CATHARTICS (some contain strychnine) — large quantity warm water, induce vomiting; Universal Antidote.

CAUSTIC LIME — do NOT induce vomiting. Give grapefruit juice, or 1 part vinegar to 3 parts water. Follow with milk.

CAUSTIC SODA — do NOT induce vomiting. Give grapefruit juice, or 1 part vinegar to 3 parts water. Follow with milk.

CHLOROFORM — baking soda solution and milk, induce vomiting.

COAL OIL — large quantity warm water, induce vomiting; Universal Antidote.

COUGH SYRUP — large quantity warm water, induce vomiting.

DDT — large quantity warm water, induce vomiting; Universal Antidote.

DIGITALIS — strong tea or tannin; Universal Antidote, induce vomiting.

DRAIN CLEANER — do NOT induce vomiting. Give grapefruit juice, or 1 part vinegar to 3 parts water. Follow with milk.

EYE DROPS — strong tea or tannin; Universal Antidote, induce vomiting.

ETHER — baking soda solution, induce vomiting. Follow with milk.

FLY SPRAY (containing arsenic) — large quantity warm salt water, induce vomiting. Follow with large dose castor oil and milk.

FLY SPRAY (containing DDT or chlordane) — Universal Antidote, induce vomiting.

GASOLINE — large quantity warm water, induce vomiting; Universal Antidote.

GOPHER POISON (containing arsenic) — large quantity warm salt water, induce vomiting. Follow with large dose castor oil and milk.

GOPHER POISON (containing cyanide) — large quantity warm water, induce vomiting.

GOPHER POISON (containing strychnine) — large quantity salt water or mustard solution, induce vomiting.

HYDROCHLORIC ACID — do NOT induce vomiting. Give water, milk of magnesia, or white of eggs beaten with water.

INK — large quantity warm water, induce vomiting.

INSECT SPRAY (containing DDT or chlordane) Universal Antidote, induce vomiting.

INSECT SPRAY (containing arsenic) — large quantity warm salt water, induce vomiting. Follow with large dose castor oil and milk.

IODINE — bread or egg whites; flour and water, induce vomiting. Follow with milk.

KEROSENE — large quantity warm water, induce vomiting; Universal Antidote.

LAXATIVES — large quantity warm water, induce vomiting; Universal Antidote.

LIME, CAUSTIC — do NOT induce vomiting. Give grapefruit juice, or 1 part vinegar to 3 parts water. Follow with milk.

LINIMENT — large quantity warm water, induce vomiting; Universal Antidote.

LYE — do NOT induce vomiting. Give grapefruit juice, or 1 part vinegar to 3 parts water. Follow with milk.

LYSOL — do NOT induce vomiting. Soapsuds, or milk and egg whites.

MATCHES — not poisonous.

MOTHBALLS (containing camphor or naphthalene) — Universal Antidote, induce vomiting.

MOTHBALLS (containing paradichlorobenzene) — not poisonous.

MURIATIC ACID — do NOT induce vomiting. Give water, milk of magnesia, or white of eggs beaten with water.

MUSHROOMS — Universal Antidote, induce vomiting.

NAPHTHALENE — Universal Antidote, induce vomiting.

NICOTINE — strong tea or tannin, induce vomiting.

NITRIC ACID — do NOT induce vomiting. Give water, milk of magnesia, or white of eggs beaten with water.

OIL OF WINTERGREEN — Universal Antidote, induce vomiting.

OXALIC ACID — large quantity lime water or chalk solution with milk and egg white, induce vomiting.

PAINT — large quantity warm water, induce vomiting.

PARIS GREEN — large quantity warm salt water, induce vomiting. Follow with large dose castor oil and milk.

PHENOL — large quantity warm water containing epsom salts, induce vomiting.

PHOSPHORUS — large quantity of milk, induce vomiting. Follow with large dose epsom salts.

PLANT SPRAY (containing arsenic) — large quantity warm salt water, induce vomiting. Follow with large dose castor oil and milk.

PLANT SPRAY (containing DDT or chlordane) — Universal Antidote, induce vomiting.

PLANT SPRAY (containing nicotine) — strong tea or tannin, induce vomiting.

POTASH — do NOT induce vomiting. Give grapefruit juice or 1 part vinegar to 3 parts water. Follow with milk.

QUICKLIME — do NOT induce vomiting. Give grapefruit juice or 1 part vinegar to 3 parts water. Follow with milk.

RAT POISON (containing arsenic) — large quantity warm salt water, induce vomiting. Follow with large dose castor oil and milk.

RAT POISON (containing phosphorus) — large quantity milk, induce vomiting. Follow with large dose of epsom salts.

ROACH POISON (containing phosphorus) — large quantity milk, induce vomiting. Follow with large dose of epsom salts.

RUBBING ALCOHOL — large quantity warm water, induce vomiting.

SILVER NITRATE — large quantity salt-water solution, induce vomiting. Follow with eggs and milk.

SLEEPING PILLS — large quantity warm water, induce vomiting. Follow with strong coffee.

SODA, CAUSTIC — do NOT induce vomiting. Give grapefruit juice, or 1 part vinegar to 3 parts water. Follow with milk.

SODA, WASHING — do NOT induce vomiting. Give grapefruit juice, or 1 part vinegar to 3 parts water. Follow with milk.

STRYCHNINE — salt water solution or mustard solution, induce vomiting.

TOADSTOOLS — Universal Antidote, induce vomiting.

TOILET BOWL CLEANER (containing sodium acid sulphate) — do NOT induce vomiting. Give 1 teaspoonful magnesia, chalk, or small pieces of soap in milk.

TURPENTINE — large quantity warm water, induce vomiting. Follow with milk.

WASHING SODA — do NOT induce vomiting. Give grapefruit juice, or 1 part vinegar to 3 parts water. Follow with milk.

WEED KILLER (containing Paris Green) — large quantity warm salt water, induce vomiting. Follow with large dose castor oil and milk.

THIS CHART MAY NOT BE REPRODUCED, ALTERED, OR SOLD FOR PROFIT EXCEPT BY PERMISSION OF EL MONTE COMMUNITY SAFETY COUNCIL, EL MONTE, CALIFORNIA

Exposure of Children to Lead

(From page 23)

the question arises: Why is lead intoxication not more prevalent than the numbers of cases presently recognized would indicate? Many cases of lesser degrees of intoxication may go unrecognized unless sought out (as indicated by the incidence of "secondary cases") or may be the result of lesser ingestion. In still other children the duration of ingestion may be insufficient to produce overt signs of lead intoxication.

However, personal-social factors elicited in those subjects studied by the authors may, in part, account for the relatively greater ingestion of lead in the subjects of this report as compared with the larger population of children who may live in similar physical environments. With respect to maternal information concerning plumbism, mothers were asked if they were aware of their child's ingestion of paint or had heard of the danger of lead poisoning. Significant is the fact that 14 of 33 mothers, while aware of the danger involved, were not concerned because they considered the amount of paint flakes their child was eating to be insufficient "to hurt him." Such maternal statements are quite understandable in view of the preceding calculation that less than 1 gm. of paint flakes may contain highly toxic quantities of lead. It is the repetitive ingestion of these minute quantities of such paint chips which leads to lead intoxication. If it is conceded that no mother can reasonably be expected to prevent this type of ingestion in the face of crumbling paint and plaster, it will be recognized that proper maintenance of both interior and exterior painted surfaces is probably the most important environmental factor in the long-term aspect of prevention of childhood lead intoxication.

The corrective efforts taken by the parents who attempted to curb the ingestion consisted largely of punitive measures which may be said, prima facie, to have been ineffectual. Only one had attempted to remove the source of lead. Common, likewise, was the lack of close parental supervision of the affected children. Thirteen of the 33 mothers were wage earners who worked at least several days of each week away from home. While away from home they left their small children with neighbors or in the care of older children. Six of the mothers had more than 3 children less than 5 years of age. They stated that in order to get various types of housework done, they shut the children in other rooms for short intervals; in these rooms sources of lead were found.

Several of the children in the hospital demonstrated an excessive desire for affection. From the point of view of the child, the ingestion of foreign material was an obvious attention-getting device in some, particularly in those who had been punished for it. Many others appeared to nibble inadvertently upon the window-sills and door frames as they gazed out into the street. In these, boredom was apparently conducive to the lead ingestion and may be attributable to the lack of an emotionally and intellectually stimulating environment. Many parents reacted by an overprotective attitude following their child's recovery from acute lead encephalopathy. They frequently sought advice about play activities for their child; the diminution in pica was often striking.

Summary and Conclusions

A study of some environmental, behavioral, and social factors in the production of lead poisoning in children in an urban community has been reported.

Most significant among the environmental data presented were the magnitude of the exposure to lead from repetitive ingestion of small quantities of leaded-paint flakes and the role of the seasonal factor (summer) in the precipitation of acute lead encephalopathy. . . . It was concluded that a child without symptoms but having increased absorption of lead, recognized during the summer months, may progress to severe encephalopathy within a few weeks if the sources of lead are not promptly identified and eliminated.

The present studies demonstrate that exposure to lead may be intense in children over 1 year of age living in dilapidated dwellings in which flaking leaded-paint is readily accessible. . . .

The importance of continued environmental exposure to lead in increasing the incidence of severe permanent damage to the brain among survivors of an initial attack of acute lead encephalopathy was demonstrated. The correlation between the occurrence of such sequelae and reexposure to lead in patients recovering from mild acute encephalopathy was statistically highly significant. It was concluded that removal of lead from the child's environment is the only adequate protective measure in such cases.

The high proportion of children aged 12 to 35 months with average intellectual capacity found among cases of lead intoxication and the high incidence of unsuspected cases found among 12- to 35-month-old housemates of the index cases demonstrate the importance of the environmental aspect of the problem in urban slum areas. This high incidence of "secondary cases" emphasizes the physician's obligation to examine carefully and promptly all environmental contacts under 3 years of age wherever an index case is found.

The developmental factors in the child and the social situations in the home which may intensify the ingestion of lead-containing materials were discussed. While the responsibility of parents to protect their children from environmental hazards is not denied, no mother can reasonably be expected to prevent the repetitive ingestion of a few paint chips when these are readily accessible. As lead is widely used as a paint drier, continued good maintenance of painted surfaces would appear to be more pertinent to the long-range prevention of childhood plumbism than specific limitations on the content of nonpigment lead in fresh paint. The various environmental data all point to the conclusion that, where housing has been permitted to deteriorate, exposure to lead may be of such intensity as to

outweigh such individual variables as mental retardation and emotional maladjustments in the child. Such intense exposure is a preventable hazard to normal small children, and, as such, constitutes a public health problem which may be more extensive than has heretofore been thought to exist.

Pending the wider availability of better housing, preventive measures will have to be adapted to individual home situations and to the available facilities within a given community. Basic to any preventive program are facilities for the prompt identification and removal of environmental sources of lead. . . . Equally important is a comprehensive social investigation of the home in order to evaluate the circumstances under which the child obtained toxic quantities of lead and to determine whether the identified sources of lead can be adequately removed or whether a change of dwelling is required to prevent further dangerous exposure to lead.

Acknowledgments

The authors wish to acknowledge the cooperation of the Baltimore City Health Department and of Huntington Williams, M.D., D.P.H., commissioner, who made its services freely available to us.

Miss Jean Askin, B.S., performed all of the psychometric examinations included in this report. . . .

References

11. Kaplan, E.: Personal communication to the author.

* * * * *

19. American Standard Specifications to Minimize Hazards to Children from Residual Surface Coating Materials (Z66.1-1955). American Standards Association, New York, 1955.

* * * * *

(Submitted May 23, accepted July 3, 1956.)

This work was supported by grants from the Lead Industries Association, New York, N. Y., and from the National Institutes of Health, Public Health Service, DHEW (G-3918).

ADDRESS: (J. J. C., Jr.) Baltimore City Hospital, 4940 Eastern Avenue, Baltimore 24, Md.

Statewide Poison Control in New York

State health Commissioner Herman E. Hilleboe has announced that a round-the-clock network of poison control and information centers, designed to protect New York State residents against the effects of accidental poisoning with household materials and drugs, is now in operation.

The network includes centers in Albany, Syracuse, Rochester and Buffalo, as well as a center in New York City which is operated under the direction of the New York City Department of Health.

The centers maintain 24-hour emergency service to treat cases, and 24-hour telephone service to answer queries from physicians on the ingredients of drugs and household supplies and to advise on latest treatment methods.

Each center is affiliated with a major teaching hospital, and can be reached by telephoning the affiliate hospital and asking for "Poison Control Center."

The telephone number in Albany is, Albany Hospital—8-4541; in Syracuse, City Hospital—4-3161; in Rochester, Strong Memorial Hospital—GReenfield 3-4400; in Buffalo, Children's Hospital —SUmmer 5100 and in New York City, New York City Health Department—WOrth 4-3800.

Pointing out that children, especially, often swallow toxic household materials and drugs, Doctor Hilleboe said that a substantial proportion of all accidental deaths among children aged one to four years were caused by accidental poisonings.

"The tragedy of such deaths is that almost all are preventable," he said. "The poison control centers have information on the ingredients of products likely to be swallowed, and on the proper methods of treating suspected poisonings.

"By concentrating such information in the centers, treatment of poisoning cases can be improved. The centers also serve as a focus for epidemiologic studies, and for research designed to evaluate various aspects of prevention and treatment."

They also will cooperate with state and local health departments, and other officials and voluntary agencies, in education programs seeking to prevent accidental poisoning.

Each center reports cases treated and cases where inquiries are answered by telephone, to the State Health Department. It also reports on the occurrence of new types of poisoning (not noted in available reference material).

Funds have been allocated by the department towards the operations of the upstate and New York City centers. Other budgetary provisions have been made so that each center may obtain books, reference material and other essential supplies, as needed.—*Bulletin*, vol. 10, No. 15, April 15, 1957, published by the New York State Department of Health, Albany, N. Y.

Antidotes for Poisoning

This Emergency PAK (Poison Antidote Kit) (see illustration) is intended for household use in case of accidental poisoning. The five plastic bottles contain antidotes. Part of the kit is a booklet (lower right) of directions for their use as emergency first aid which lists over 700 products which contain active and potential poisons and their antidotes. —*Olender & Reiss Laboratories, Detroit, Mich.*

Sample Evaluation Form Available From N.S.C.

Although no National Home Safety Inventory will be conducted for 1957, the Home Division will be happy to supply a sample form suitable for duplication, or the actual mimeographed questionnaire forms to any community or state desiring to conduct its own Inventory of 1957 Home Safety Activities. If such service is desired, kindly contact the Home Division by November 1, 1957.

r es-
, vol.
d by
t of

g

ison
on)
case
five
Part
ght)
ner-
700
and
otes.
ries,

ome
on-
ion
ple
the
aire
de-
ory
If
on-
em-

. 8
-107